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Differences in Internalizing Symptoms and Cognitive Functioning in Adults with Attention-Deficit/Hyperactivity Disorder and Sluggish Cognitive Tempo

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DIFFERENCES IN INTERNALIZING SYMPTOMS AND COGNITIVE FUNCTIONING IN ADULTS WITH ATTENTION-DEFICIT/HYPERACTIVITY DISORDER AND SLUGGISH COGNITIVE TEMPO

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DISSERTATION APPROVAL

This is to certify that the thesis presented to us by Avery B. Ducey on the 7th day of May, 2021, in partial fulfillment of the requirements for the degree of Doctor of Psychology, has been examined and is acceptable in both scholarship and literary quality.

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Abstract

Sluggish cognitive tempo (SCT) is a recently identified mental health construct. Currently, no widely accepted diagnostic criteria for SCT exist, and it is not recognized in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*). There is debate in the psychological community as to whether SCT is better conceptualized as an atypical presentation of attention-deficit/hyperactivity disorder (ADHD) or a unique symptom cluster comprised of ADHD and additional psychological and neurocognitive symptoms. When controlling for ADHD symptomatology, SCT has been found to be associated with internalizing symptoms, such as anxiety and depression, as well as impaired cognitive functioning, such as deficits in executive function and slow processing speed. The current study examined groups of adults diagnosed with varying levels of ADHD and SCT symptomatology to determine whether they differed in their internalizing symptoms and cognitive functioning. Analyses indicated subjects with clinical levels of both ADHD and SCT had higher scores on measures of internalizing symptoms and executive dysfunction than those with ADHD and subclinical symptoms of SCT or those with ADHD only. Regression analyses identified symptoms of depression and executive dysfunction that significantly predicted subjects SCT symptoms. It is hoped the current study will inform the assessment and treatment of adults with ADHD, SCT, and internalizing symptoms.

Chapter 1: Introduction

Statement of the Problem

The American Psychiatric Association's (2013) *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; *DSM-5*) defines attention-deficit/hyperactivity disorder (ADHD) as a neurodevelopmental disorder that results in difficulty sustaining attention, hyperactivity, and/or impulsive behavior that interferes with social, academic, or occupational functioning (American Psychiatric Association, 2013). Sluggish cognitive tempo (SCT) is a conceptualized as constellation of psychological symptoms characterized by feelings of apathy and lethargy, difficulty concentrating, daydreaming, and slowed cognition that negatively impacts mental health functioning (Becker & Barkley, 2018). Initially hypothesized as a potential subset of ADHD symptomatology, recent research has concluded that SCT is distinct from ADHD, and other mental health disorders (Barkley, 2012, 2014; Becker, Marshall, & McBurnett, 2014). Although SCT is recognized, by some, as a separate disorder from ADHD, approximately half of all adults with clinically elevated symptoms of SCT have a co-occurring ADHD diagnosis (Barkley, 2012).

A literature review of PsychINFO, Google Scholar, and Ebscohost revealed a number of studies exploring how children and adults with ADHD and SCT differ in the types of difficulties they experience. The most relevant are cited below. Studies of these differences in children and adults found unique associations of SCT with internalizing symptoms, lower academic performance, lower annual income, impaired organization and problem solving skills, increased stress, and poorer quality of life, even when

controlling for the influence of more widely-accepted ADHD symptoms (Becker et al., 2018; Becker, Marshall, & McBurnett, 2014; Barkley, 2012; Combs et al., 2014, 2015).

Although SCT and ADHD are theorized to be distinct from one another, their close relationship, along with SCT's status as a newly identified and less well-defined mental health construct makes SCT difficult to examine. At this point, it is unclear whether SCT is a transdiagnostic factor present in many psychopathologies or is better conceptualized as a distinct mental health issue or diagnosis (Lunsford-Avery & Mitchell, 2018). Few studies have examined both ADHD and SCT's close relationship with internalizing symptoms and various types of comorbid dysfunction or impairment associated with these disorders in adult populations (Becker & Barkley, 2018).

Purpose of the Study

ADHD and SCT are two related but distinct psychological syndromes that are both associated with impairment in social-emotional, academic, vocational, and executive functioning (Becker & Barkley, 2018). SCT is still a relatively new psychological construct that is poorly understood, due to the both complexity of the phenomenon and limited research. At this point, it there is debate about whether SCT should be conceptualized as a transdiagnostic factor present in many psychopathologies or as a distinct mental health issue, which is more or less related to ADHD (Lunsford-Avery & Mitchell, 2018). A small but growing number of studies have examined both ADHD and SCT's close relationship with internalizing symptoms and differences in the types of comorbid impairment associated with these disorders in adult populations (Becker & Barkley, 2018). This study examined how comorbid issues present in adults with varying levels of SCT and ADHD. Specifically, this study sought to learn what, if any,

differences exist between these groups in their internalizing symptoms and cognitive functioning.

SCT and ADHD are distinct mental health constructs, with evidence for comorbidity. By examining the differences in internalizing symptoms and cognitive functioning in adults with varying levels of ADHD and SCT, it is hoped that this study will better inform assessment and treatment planning for these often complex and challenging cases. Gaining knowledge of these differences may improve clinicians' capacity to differentiate between SCT and ADHD during their diagnostic process as well as offer potential targets for treatment the clinician and client may not have initially considered.

Consequently, this study compared subjects who meet criteria for adult ADHD and SCT (ADHD + SCT), with subjects who meet criteria for ADHD with subclinical levels of SCT (ADHD + subclinical SCT), subjects who only meet criteria for ADHD (ADHD only), and subjects who only meet criteria for SCT (SCT only). These diagnostic groups were compared on measures of internalizing symptoms and cognitive functioning. In this way, the four groups could be compared to determine if there were significantly different levels of internalizing symptoms and cognitive functioning. Furthermore, this study analyzed subjects' data to identify which variables are the best predictors of SCT symptomatology.

Hypotheses

Hypothesis 1. There would be a significant difference between subjects diagnosed with ADHD + SCT, ADHD + subclinical SCT, ADHD only, and SCT only, with those diagnosed with ADHD + SCT having higher levels of internalizing symptoms

than the other groups. For hypothesis 1 and hypothesis 2, ADHD was operationally defined as meeting *DSM-5* criteria for ADHD-In, ADHD-H/I, or ADHD-C as determined by clinician ratings from the Structured Clinical Interview for *DSM-5* Disorders (SCID-5). SCT was operationally defined as a score of 93rd percentile or higher for the subjects' reference age group on the BAARS-IV SCT subscale (the threshold for clinically significant levels of SCT outlined in the BAARS-IV manual). Subjects whose SCT scores are in the Borderline range as outlined in the BAARS-IV manual (85th to 92nd percentile) do not meet criteria for SCT while still having notable levels SCT symptomatology. Their borderline impaired scores do not allow SCT to be easily classified as present or absent. This presentation of SCT is referred to as "subclinical SCT" for comprehension's sake. Subjects were considered to not meet criteria for SCT if their scores on the BAARS-IV SCT subscale were at the 84th Percentile or lower (reflecting nonsignificant or marginal levels of SCT as outlined in the BAARS-IV manual). Internalizing symptoms were operationally defined as scores on the BDI-II, Penn State Worry Questionnaire (PSWQ), the affect subscale of the Brown Attention Deficit Disorder Scale-Adult Version (BADDS), and Neuroticism scale of the NEO-PI-R.

Hypothesis 2. There would be a significant difference between subjects diagnosed with ADHD + SCT, ADHD + subclinical SCT, ADHD only, and SCT only, with those diagnosed with ADHD + SCT exhibiting significantly more impaired cognitive functioning than all other groups. Cognitive functioning was operationally defined as subjects' performance on the Coding task and Digit Span task of the WAIS-IV, and total executive functioning summary score of the BDEFS.

Hypothesis 3. Anxiety and deficits in executive functioning will predict the level of SCT after accounting for ADHD symptomatology. SCT was operationally defined as subjects' percentile rank on the BAARS-IV SCT subscale (with 93rd percentile and above representing clinically significant levels of SCT). Anxiety was operationally defined as subjects' scores on the PSWQ. Executive functioning was operationalized as subjects' total executive functioning summary score of the BDEFS. ADHD symptomatology was operationally defined as subjects' scores on the *DSM-IV* Inattentive symptoms and *DSM-IV* Hyperactive/Impulsive symptoms scales of the CAARS.

Chapter 2: Review of the Literature

Attention-Deficit/Hyperactivity Disorder in Adults

ADHD is typically thought of as a disorder of childhood, but the identification of a 4.4% prevalence of the disorder in adults has been gaining increasing attention among clinicians and researchers in recent years (Kessler et al., 2006). The *DSM-5* uses two symptom dimensions, inattention and hyperactivity/impulsivity, to identify three recognized subtypes of ADHD: predominantly inattentive type (ADHD-In), predominantly hyperactive-impulsive type (ADHD-H/I), and the combined type (ADHD-C) (American Psychiatric Association, 2013). The combined subtype is the most common in adults, with approximately 62% of adults exhibiting ADHD-C, 31% exhibiting ADHD-In, and 7% exhibiting ADHD-H/I. The prevalence of ADHD-C and ADHD-In in adults indicates that upwards of 90% of those with adult ADHD exhibit clinically significant levels of inattentive symptoms (Wilens et al., 2009). These rates indicate that the majority of adults diagnosed with ADHD experience symptoms from the inattentive domain of ADHD. Several symptoms of ADHD in adults are nonspecific and can stem from other forms of neurological impairment or difficulties caused by another mental health issue.

ADHD is highly comorbid with many other mental health disorders, including anxiety disorders, mood disorders, substance use disorders, and intermittent explosive disorder (Kessler, et al., 2006). Individuals with adult ADHD have a higher lifetime comorbidity rate of internalizing disorders and externalizing disorders compared to the general population. These include internalizing disorders such as depressive disorders, anxiety disorders, and cluster C personality disorders; as well as externalizing disorders

such as Cluster B personality disorders, conduct disorder, and oppositional defiant disorder (Jacob et al., 2014). High levels of effortful control and low stress levels are protective factors against internalizing disorders (Gulley et al., 2017).

Models of Attention and Executive Functioning.

Deficits in attention and self-control (a lack of inhibition) are central to ADHD symptomatology. Many models of attention and self-control attribute the symptoms of ADHD and SCT to a deficiency or failure of neuropsychological processes. This failure has a downstream effect on the capacity to self-regulate behavior.

An earlier model of ADHD, the self-regulation model, posited by Russell Barkley (1997) conceptualizes ADHD as the expression of severely impaired inhibition. In his model Barkley defines inhibition as "performance on cognitive and behavioral tasks that require withholding of responding, delayed responding, cessation of ongoing responses, and resisting distraction or disruption by competing events" (Barkley, 1997, p. 68). When the cognitive processes that control inhibition are impaired or dysregulated, cognition becomes overburdened by an unfiltered stream of stimuli and individuals are less capable of self-regulating responses to said stimuli. Deficits in inhibition lead to thoughts and behaviors being influenced more by immediate rewards than by long-term goals or executive functioning (Barkley, 1997). The degree to which one is capable of resisting stimuli irrelevant to their current goal is defined as persistence. The weaker or more dysfunctional the executive functions that control inhibition, the less persistent one is in tasks, resulting in the hyperactive/impulsive and inattentive behavior characteristics of ADHD (Barkley, 1997; Ducey, 2016).

Barkley's more recent self-regulation model belongs to the executive function theory of ADHD. Broadly speaking, executive functioning is a set of top-down mental abilities that requires the collaboration of different cognitive and neuropsychological processes. These processes work together to help an individual monitor and regulate one's own behavior over time towards the achievement of a goal or to solve a novel problem. When a person's mental abilities are insufficient to adequately monitor and regulate one's own behavior and as a result, the individual is having difficulty attaining a goal or solving a novel problem, they are said to be experiencing executive dysfunction. Multiple researchers have proposed that deficits in a specific executive function or broad deficits in executive functioning are the source of the inattentive, hyperactive, and impulsive symptoms of ADHD (Willcutt et al., 2005). Research consistently finds significant relationships and moderate effect sizes in executive functioning deficits and ADHD symptomatology. The most consistent executive functioning deficits related to ADHD are in inhibition, vigilance/sustained attention, working memory, and planning (Willcutt et al., 2005).

Another model of attention difficulties in a variety of disorders is Posner's model of attention. Posner's model posits that separate neurocognitive systems are in charge of alerting responses to the appearance of new stimuli; orienting to said stimuli; and executive control of thoughts, actions, and emotions in response to these stimuli (Becker & Willcutt, 2018). Some have theorized that deficiencies in the orienting system may be the cause of SCT symptomatology whereas ADHD symptoms represent deficiencies in all three systems (Barkley, 2016; Becker & Willcutt, 2018).

Sluggish Cognitive Tempo

Validating SCT.

In the 1980s, the delineation between the two symptom dimensions of ADHD (then known as Attention Deficit Disorder; APA, 1987) was codified into the third edition of the *Diagnostic and Statistical Manual of Mental Disorders.* This change was made to account for children who presented with inattentive but not hyperactive symptoms of ADHD. Following the release of the *DSM-III,* researchers set out to examine the differences between these two symptom domains.

The first of several factor analytic studies examining the symptoms and related behaviors of children with ADHD examined the factors that emerged from behavior rating scales of children with ADHD (Neeper & Lahey, 1986). They found that a three-factor model provided the best fit for ADHD. In addition to the expected inattentive and hyperactive factors, a "slow tempo" factor emerged. Across multiple studies of ADHD symptoms and related behaviors in children, these three factors: hyperactivity–impulsivity, inattention–disorganization, and "slow tempo" have emerged (Bauermeister et al., 2012; Becker, Marshall, & McBurnett, 2014). As research progressed, researchers grouped the constellation of symptoms and behaviors related to the slow tempo factor under the label sluggish cognitive tempo (SCT).

The SCT factor consisted of items that captured behaviors described as sluggish, apathetic, lethargic, drowsy, and being "in a world of his or her own." Another factor analytic study found 13 features that showed good factor loadings with SCT. This included sluggishness, being tired or lethargic, slow thinking/processing, loses train of thoughts easily, sleepy or drowsy, spacey, daydreaming, being "in a fog," being

underactive or slow moving, getting lost in thought, staring blankly into space, easily confused, and being apathetic or unmotivated. Further studies showed that SCT symptoms were uniquely elevated in children with ADHD-In compared to ADHD-H/I. (Becker, Marshall, & McBurnett, 2014). Studies of children from general population and clinical samples identified the presence of SCT in children without clinical levels of ADHD symptomatology and found SCT symptoms to be associated with inattentive symptoms (Garner et al., 2010; Wåhlstedt & Bohlin, 2010).

In 1987, changes in the revised edition of the DSM-III eliminated the new practice of diagnosing ADHD subtypes (APA, 1987). This had the inadvertent effect of exiling SCT from entering the official nosology used to diagnose ADHD (Becker, Marshall, & McBurnett, 2014). In spite of increased research interest in this third dimension of ADHD and evidence that SCT represented a distinct and important domain of attentional problems in those with ADHD, SCT remains unincorporated into the official nosology of ADHD to this day (Becker, Marshall, & McBurnett, 2014). The current conceptualization of SCT describes it as a set of symptoms characterized by feelings of apathy and lethargy, difficulty concentrating, and slow cognitive processing speed, with strong relationships to established ADHD symptom dimensions (Becker & Barkley, 2018).

As clinical and research interest in adult ADHD grew, interest in whether SCT presented in adults followed close behind. Several comprehensive studies were conducted by Dr. Russell Barkley as part of the development of the fourth edition of the Barkley Adult ADHD Ratings Scale (BAARS-IV). The BAARS-IV is a self- and other-report instrument designed to measure the inattentive, hyperactive, and impulsive symptoms of

ADHD in adults ages 18-89. For the fourth edition, -nine items used in prior studies of SCT in children were added to the BAARS-IV measure. These SCT items and other items on the BAARS-IV used to assess for ADHD symptomatology in adults were administered to a normative sample of the general population consisting of 1,249 adults in the United States. Barkley proposed that an individual must score higher than the 93rd percentile of the adults in the sample within the same age bracket on the SCT subscale of the BAARS-IV before they are considered to have clinically significant levels of SCT (Barkley, 2011a). The SCT subscale measures chronic daydreaming, hypoactivity, mental "fogginess," and difficulty sustaining concentration and alertness during boring tasks.

Later studies by Barkley used the same normative sample of the general population in the Unites States to conduct multiple factor analyses examining differences between adults whose SCT scores were in the 95th percentile or higher of the normative sample with those whose ADHD scores were in the 95th percentile or higher. This research confirmed that the chronic daydreaming, hypoactivity, mental fogginess, and tendency to become bored easily, characteristic of SCT, aligns with a different factor than the hyperactive-impulsive and inattentive dimensions of ADHD (Barkley, 2011a).

Barkley determined that, of the participants who met the criteria for clinical levels of SCT, 54% also met the criteria for a diagnosis of ADHD. Similarly, 46% of those diagnosed with ADHD also met criteria for clinical levels of SCT. This overlap between the two disorders is attributed to participants with SCT and/or ADHD both sharing high levels of inattentive symptoms (Barkley, 2012). SCT symptoms shared as much as 50% of their variance with inattentive symptoms but less than 25% of the variance with hyperactive/impulsive symptoms. Furthermore, SCT scores were associated with

impairment in social, academic, and emotional functioning, even after controlling for comorbid ADHD symptomatology (Becker & Barkley, 2018). Based on these findings, Barkley concluded elevated SCT symptoms likely reflect a separate disorder from ADHD that is comorbid in approximately half of adults with ADHD (Barkley, 2012).

Another study set out to examine the reliability and validity of SCT measures in an applied clinical setting. Lunsford-Avery et al. (2018) examined client data from a medical center's outpatient specialty clinic for ADHD. This data was collected as part of the initial psychological evaluations of new clients presenting for services at the program. They found the SCT subscale on the BAARS-IV to be reliable and valid for use in this outpatient clinical setting.

Exploratory factor analysis also found the SCT items of the BAARS-IV clustered into three factors: sleepy/sluggish, low initiation/persistence, and slow/daydreamy. The low initiation/persistence and slow/daydreamy factors were significantly correlated with both inattentive and hyperactive/impulsive symptoms of ADHD. The sleepy/sluggish factor was positively associated with inattention but not hyperactivity/impulsivity (Lunsford-Avery et al., 2018).

Empirical studies with large samples of adults and children in a variety of settings have demonstrated that SCT is a distinct psychological construct, which is comorbid in approximately half of adults with ADHD, particularly so in inattentive presentations (Barkley, 2011a, 2012; Becker et al., 2016; Garner et al., 2010; Wåhlstedt & Bohlin, 2010). The development and application of measures for SCT have made it possible to measure SCT in research participants, clinical samples, and the general population (Barkley, 2011a, 2012; Lunsford-Avery et al., 2018). Researchers are able to examine the

subtle differences in how SCT and ADHD relate to each other, in addition to other mental health disorders.

Depletion Models and SCT.

The resource model of control posits that the ability to -control one's behavior including thoughts, emotions, and actions relies on limited resources that are depleted as an individual exercises self-control. This model defines self-control as a "resource" of ability or that allows an individual to attain personal goals by choosing larger but delayed long-term rewards over smaller but immediate sources of gratification. As this resource is depleted, it becomes more and more difficult for the individual to monitor and self-control their behavior, resulting in a state referred to as "ego-depletion" (Baumeister, 2002). Whereas the original theory posited that blood glucose might be the resource depleted by this process, later research demonstrated this was not the case (Gailliot et al., 2007; Inzlicht & Schmeichel, 2012).

Inzlicht and Schmeichel proposed a revised version of the resource model. Their process model of depletion argues that exerting self-control influences motivation and attention, which increases the likelihood of a failure in self-control. As mental effort is exerted to maintain self-control, motivation begins to shift away from the goal or reason for expending effort to maintain self-control (e.g., motivated to maintain a diet) and towards a more immediate source of gratification (e.g., motivated to eat ice cream). This shift in motivation is coupled with a shift in attention. As one exerts effort to maintain self-control, attention shifts away from stimuli or information that they need to continue to exert self-control (e.g. focusing on how eating ice cream conflicts with a dieting goal) and towards potentially rewarding stimuli (e.g. focusing on how delicious ice cream

looks). Inzlicht and Schmeichel proposed ways to counteract the ego-depletion effect, such as presenting cues to help refocus attention on reasons for self-control and providing rewards or incentives for sustaining self-control, which would increase motivation

A study examining the ego-depletion paradigm in adults with ADHD compared participants with a history of ADHD and a control group without ADHD on their performance on a handgrip stamina task and computerized gambling task following a continuous performance task. Continuous performance tasks require prolonged, sustained attention and are notoriously frustrating and boring. They are thought to be a good way to induce ego depletion and measure sustained attention in a laboratory setting. The handgrip stamina task and computerized gambling task were used to operationalize self-control for this study (Lubusko, 2005).

The continuous performance task was administered to both groups to cause ego-depletion. Participants' performance on the gambling task and handgrip task were then measured and compared. Results indicated no differences between the participants with ADHD and without ADHD on their performance on the gambling task and handgrip task following the continuous performance task. This indicates that there was no observed difference between the participants with ADHD and without ADHD in the degree of ego-depletion they experienced (Lubusko, 2005).

Although these findings do not support the idea of more severe ego-depletion in adults with ADHD, it is important to consider alternative models of resource depletion and limitations to the design of the study. This study was conducted before Inzlicht and Schmeichel published their model. Considering these findings in the light of the process model of depletion, the participants were first given a task that potentially depleted their

attention and motivation, then showed no between groups differences on measures of impulsivity, persistence, and decision making. The continuous performance task may have decreased participants' attention and motivation, but changes in attention and motivation were not measured in this study. The gambling task was originally designed to measure decision-making and impulsivity and the handgrip task was designed to measure persistence and physical stamina (Lubusko, 2005). Retrospective consideration of these findings indicates that the researchers may have erroneously used measures of impulsivity and physical exertion to examine the depletion of self-control.

Individuals with ADHD struggle to regulate attention, sustain motivation, exhibit self-control, and activate other executive functions (American Psychiatric Association, 2013; Barkley, 2011b; Willcutt et al., 2005). If attention and motivation play a key role in sustaining self-control as posited by the process model of depletion, then those with ADHD would have a particularly difficult time exhibiting self-control due to their impaired attention and motivation processes, not just because of depletion of a general self-control resource.

A study comparing children with ADHD and typically developing children on a continuous performance task found modest evidence for this model. A continuous performance task was administered to all participants twice: once to cause ego-depletion, induced by prolonged sustained attention to boring stimuli, and a second to measure performance. Half of the children with ADHD and half of the typically developing children were offered token reinforces for good performance on the continuous performance task that could be exchanged for a small toy at the end of the experiment (Dekkers et al., 2017).

The children with ADHD exhibited significantly more omission errors and greater reaction time variability, both of which are measures of basic attention and common in ADHD (Dekkers et al., 2017), than the control group on the initial continuous performance task. The ADHD and control groups did not differ significantly on measures of inhibition or reaction time (measures of self-regulation) on the initial task. Children with ADHD and typically developing children who were offered reinforcement for their performance on the second administration of the task all showed less reaction time variability and fewer omission errors than those who were not. Furthermore, participants with ADHD who were offered reinforcement performed better than those with ADHD who were not given reinforcement for their performance. Reinforcement effects did not differ between children with ADHD and typically developing children. One potential explanation for the lack of difference in reinforcement effects put forth by the authors is the elevated reward threshold in children with ADHD. The inexpensive toy might have been an insufficient reward to instill high levels of motivation in children with ADHD, but was a sufficient reward to produce moderate levels of motivation found in typically developing children (Dekkers et al., 2017).

The results of this study suggest ego-depletion had an impact on measures of basic attention but not self-regulation. This decrease in attention was more prevalent in children with ADHD, possibly due to their already impaired capacity to sustain attention. The ego-depletion effect on attention was mitigated by increasing motivation through an external reward, and this mitigating effect brought the performance of children with ADHD to the same level as typically developing children (Dekkers et al., 2017). The original resource model of control and the notion of ego-depletion have come under

scrutiny due to several conflicting meta-analyses and replication studies (Hagger et al., 2016; Pollert, 2015). However, these findings suggest that the process model of depletion may accurately describe the relationship between sustained effort, motivation, and attention in those with impaired executive functioning, including those with ADHD and potentially SCT. To date, a literature review reveals no studies specifically examining SCT in relation to the resource model of control or process model of depletion.

Nosology and Transdiagnostic Factors

The *DSM-5* conceptualizes mental health disorders in categorical terms, requiring a diagnostic threshold of symptoms and other criteria before diagnostic criteria are met for various disorders. The *DSM-5* classifies disorders with similar features, etiology, and diagnostic criteria into categories of related disorders; for instance, neurodevelopmental disorders or psychotic disorders. In the past, disorders in different categories were presumed to have independent etiologies and share few features in common (American Psychiatric Association, 2013). Whereas this framework makes psychopathology easier to diagnose and classify, it is recognized that "the boundaries between many disorder 'categories' are more fluid over the life course . . . and many symptoms assigned to a single disorder may occur, at varying levels of severity, in many other disorders," (American Psychiatric Association, 2013, p. 5). Furthermore, individuals with a mental health disorder are more likely to present with multiple diagnoses than with a single disorder (Kessler et al., 2005). Additionally, those who present with symptoms that are subthreshold to the diagnostic criteria of a disorder can still experience clinically significant distress and impairment in their lives (Krueger & Eaton, 2015). The high

prevalence of comorbidity and overlap of symptomatology in various mental health diagnoses indicated that there might be common factors shared between many disorders.

Internalizing and Externalizing Factors

According to the theory of transdiagnostic factors (TDFs), many psychological disorders with similar features (such as disorders of anxiety and mood) are hypothesized to stem from one or two underlying core problems, in this case, internalization and externalization of negative affect. When individuals experiences distress or is attempting to cope with negative emotion, they can choose to internalize these problems by keeping these feelings inside or, otherwise, not acknowledging how they feel. They could also externalize their problems and direct these negative feelings away from themselves by acting out towards people or objects in their environment. According to this theory, these underlying factors express themselves as different disorders depending on each individual's unique characteristics and experiences (Krueger & Eaton, 2015).

One of the first and most prominent theories of TDFs posited the distinction between internalizing and externalizing factors in mental health disorders (Achenbach, 1966). A factor analytic study of psychopathology in children with a variety of mental health disorders identified that their symptoms broadly loaded onto these two factors: internalizing and externalizing (Achenbach, 1966). The internalizing factor consisted of symptoms and behaviors directed inwards and influence the individual's internal psychological experience, such as anxiety, depressive symptoms, social withdrawal and isolation, somatic complaints, and traumatic stress. The externalizing factor consisted of symptoms and behaviors directed outwards on the individual's environment, such as disruptive, hyperactive, and aggressive behaviors. (Jacob et al., 2014). Later research has

validated the existence of these externalizing and internalizing factors in a variety of populations (Krueger & Eaton, 2015).

Additional TDFs have been proposed representing common factors to a large number of psychopathologies, which do not load onto the externalizing or internalizing factors. These include, but are not limited to, a thought disorder factor, which is common to schizophrenia and other psychotic disorders, and an autism factor, which is unique to individuals across the autism spectrum (Krueger & Eaton, 2015). Others contend that even these factors could be attributed to internalizing or externalizing factors. Following the identification of SCT in the mid-1980s, subsequent research has investigated whether SCT is another subtype of ADHD, a distinct mental health disorder, or a TDF that contributes to multiple mental health disorders.

There is currently no consensus regarding the core constructs or nosology used as a comprehensive definition of SCT (Becker et al., 2016). A meta-analysis evaluating the validity of SCT as a diagnostic construct identified 18 potential core features of SCT across multiple disparate measures used by researchers to measure SCT. Of these 18 core features, only 13 showed good factor loadings onto the SCT factor in this meta-analysis, and encompassed the cognitive and behavioral features of SCT (Becker et al., 2016). The other five domains had insufficient factor loadings or loaded stronger onto other factors such as ADHD-In symptoms or depression. The SCT construct demonstrated good internal validity and moderate external validity across the studies examined. Moderate and significant correlations were found between SCT ratings and nearly all of the other psychopathologies examined in this study. However, none of these associations were high enough that SCT could be considered a redundant conceptualization of a preexisting

psychiatric disorder, supporting the notion that SCT is a separate construct from other better established and defined established psychopathologies. However, to date, there is still insufficient research on SCT to determine if it is best conceptualized as an independent psychiatric disorder or better understood as a syndrome or TDF that is present across many pathologies (Becker et al., 2016).

Internalizing Disorders in Those With SCT and ADHD

A handful of studies have investigated the relationships between SCT; the inattentive, hyperactive, and impulsive presentations of ADHD; and internalizing disorders. Symptoms of internalizing disorders include anxiety, depression, social withdrawal, isolation, somatic complaints, and traumatic stress. Anxious and depressive disorders are two of the most common mental health issues in the population. A national survey by the American Medical Association examined the prevalence of mental health diagnoses from the *DSM-IV* in the United States. They found anxiety disorders to be the most prevalent class of disorders, with 28.8% of the population sample meeting criteria for an anxiety disorder at some point in their life. More specifically, the survey also found that the four most prevalent mental health disorders were major depressive disorder (16.6% prevalence), alcohol abuse (13.2%), specific phobia (12.5%), and social phobia/social anxiety disorder (12.1%) (Kessler, Berglund, Demler, Jin, Merikangas, & Walters, 2005).

Anxiety disorders are characterized by excessive fear and anxiety, leading to behavioral disturbances. Fear is the negative emotional response to a real, imminent threat to one's well-being, whereas anxiety is the anticipation of future threat (American Psychiatric Association, 2013). Anxious symptoms can occur in response to real threats

to an individual, and typically dissipates after the threat is gone. Anxiety is considered pathological when it regularly occurs in response to perceived, rather than actual threats or is disproportionate to the actual threat posed to the individual. Depressive disorders are characterized by sadness, irritability, and feelings of emptiness, that are accompanied by somatic and cognitive changes. These depressive symptoms significantly impair functioning (American Psychiatric Association, 2013).

Studies of SCT in adults and children have found it to be significantly associated with internalizing symptoms including social withdrawal, somatic complaints, anxious symptoms, and depressive symptoms (Bauermeister et al., 2012; Becker, Langberg, et al., 2014). Studies examining externalizing symptoms have found them to be unassociated or negatively associated with SCT when controlling for the influence of ADHD symptoms (Becker, Marshall, & McBurnett, 2014; Becker, & Langberg, 2013). These findings illustrate that SCT and ADHD each have a unique relationship with internalizing symptoms, whereas externalizing symptoms are related to ADHD but not SCT.

This research has established that both ADHD and SCT are related to anxiety and depression. However, the close relationship between ADHD and SCT warranted examination as to whether the relationship between anxious and depressive symptoms and ADHD could be attributable to SCT or vice versa. An additional layer of difficulty comes from the challenge of measuring symptoms of anxiety in those with the hyperactive symptoms of ADHD, whose chronic restlessness and higher levels of physiological arousal can be mistaken for anxiety (Meyer, Miller, Metzger & Borkovec, 1990; Ramsay, 2015). Furthermore, individuals with ADHD may underreport their level

of impairment on self-report measures due to a lack of self-awareness on their behavior (Manor et al., 2012).

One study examining ADHD, SCT, and internalizing symptoms examined the symptom profiles of 2,744 children diagnosed with the Inattentive or Combined subtype of ADHD. Their information was collected as part of a larger study examining the effectiveness of mental health service delivery across nine predominantly low-income school districts in Texas. Children with ADHD-In were sorted into a high SCT or low SCT group based on whether they had elevated scores on two items from a teacher rating scale examining the SCT symptoms of "daydreams or gets lost in his/her thoughts" and "underactive, slow moving, or lacks energy" (Carlson, & Mann, 2002).

These students were also administered the teacher rating form of the Adjustment and Behavior Problems Scales (TRF-ABPS), a standardized measure of various childhood behavior problems and functioning rooted in Achenbach's theory of internalizing and externalizing disorders. The TRF-ABPS includes an overall internalizing behavior composite score calculated from subscales examining symptoms such as acting withdrawn, somatic complaints, anxious/depressed symptoms, and social problems. The TRF-ABPS also includes an overall externalizing symptoms composite score consisting of subscales examining delinquency and aggressive behavior (Achenbach, 1991).

Pairwise analyses of variance revealed teachers rated children in the high SCT/In group significantly higher on the internalizing subscales of overall internalizing behavior, acting withdrawn, and somatic complaints on the TRF than those in the low SCT/In group and those in the ADHD-C group. Children in the high SCT/In group were rated

significantly lower on the externalizing subscale of aggressive behaviors than those in the low SCT/In group and those in the ADHD-C group. Although this study was severely limited by failure to control for SCT levels in the ADHD-C group and the use of only two items from a teacher rating scale to measure SCT, it is one of the first to demonstrate that children with ADHD and SCT can be distinguished from those with only ADHD based on co-occurring mental health symptoms (Carlson & Mann, 2002).

A series of additional studies examining the relationship between SCT, ADHD, anxiety, and depression in adults utilized a nonclinical sample of undergraduate students, ranging in age from 17 to 34. Hierarchical linear regression examined SCT's relationship with anxiety and depression and academic adjustment while controlling for demographic variables and the inattentive, hyperactive, and impulsive symptoms of ADHD (Becker, Langberg, et al., 2014). The Depression Anxiety Stress Scales (DASS-21) were used to assess the degree of depression and anxiety participants had experienced in the past week. Previous studies of the DASS-21 have demonstrated its internal consistency and concurrent validity with other measures of depression and anxiety (Antony, Bieling, Cox, Enns, & Swinson, 1998).

Preliminary correlations were used to identify which demographic variables and ADHD symptoms would be appropriate to use as predictors in the analyses. Comparisons were made between two regression models. The first model used the age, sex, and the inattentive, impulsive, and hyperactive subscales from the BAARS-IV as predictors, and a second model that added in the SCT subscale of the BAARS-IV as an additional predictor. These models were used to predict scores on measures of anxiety, depression, and other measures of academic success (Becker, Langberg, et al., 2014).

Comparisons between the two models' ability to predict anxiety scores on the DASS-21 found that the model which included SCT as a predictor explained 6% more of the variance than the model which excluded SCT. Furthermore, adding SCT scores as a predictor in step two reduced inattentive symptoms and impulsive symptoms ability to predict anxiety to nonsignificance. Comparisons between the two models' ability to predict depression scores on the DASS-21 found the second model including SCT explained an additional 8% of the variance compared to the first model. In the first model, inattentive and hyperactive symptoms were found to be significant predictors of depression. In the second model, only SCT emerged as a significant predictor of depressive symptoms. (Becker, Langberg, et al., 2014).

Another study in this series tested to see if SCT symptoms would continue to be the only significant predictor of depression in a sample of college students, in the same age range, who met the criteria for ADHD-In or ADHD-C. The model in step one utilized ADHD subtype as a categorical variable, age, and a continuous measure of current mental health service use. The second added the SCT subscale of the BAARS-IV. Consistent with the first study, the model including SCT explained an additional 8% of the variance explained compared to the first model ($\Delta R^2 = .08$, $\Delta F(1, 67) = 7.67$, $p < .01$). Age remained a good predictor of depression in both models, but neither ADHD subtype nor current mental health treatment usage were significant predictors of depression as measured by the DASS-21 in either model (Becker, Langberg, et al., 2014).

Another study examined whether there were differences between adults diagnosed with ADHD who were taking stimulants to treat their symptoms compared to those with adult ADHD who were unmedicated. Separate regressions were run for each group using

scores on measures of anxiety and depression, and SCT. Participants' SCT levels, as measured by the BAARS-IV, were correlated with ADHD symptomatology in both groups. (Leikauf & Solanto, 2017). The State-Trait Anxiety Inventory (STAI) measured participants' anxiety and the Beck Depression inventory (BDI-II) measured their depression. The STAI is a clinical measure examining the extent a participant is experiencing anxiety in the moment (state anxiety) and their general tendency to respond to stressful situations and events with anxiety (trait anxiety; Spielberger, 1989). The BDI-II is a widely used self-report measure designed to examine the severity of depression within the past two weeks (Beck et al., 1996). A multitude of studies have confirmed the reliability and validity of the BDI-II in measuring the severity of depressive symptomatology across different cultural groups and clinical populations (APA, 2019). The reliability and validity of the STAI as a measure of both state and trait anxiety has been verified by its creators (Spielberger et al., 1983; Spielberger, 1989).

Participants' scores on trait anxiety and state anxiety on the STAI and their scores on the BDI-II were significantly positively correlated with SCT in the unmedicated group, but were not significant in the group taking stimulants (Leikauf & Solanto, 2017). The executive functioning results of this study are discussed below. Because there were no premedication measurements of the participants, anxiety and depression levels, it is uncertain whether these findings were due solely to the effect of stimulant use or to another difference between the unmedicated group and group using stimulants (Leikauf & Solanto, 2017).

A recent study of SCT in adults with current ADHD symptomatology sorted participants into three groups, based on whether they exhibited minimal, moderate, or

severe levels of SCT as measured by the BAARS-IV. Researchers used MANOVAs to compare the three groups across their presenting levels of internalizing symptoms, anxiety, depression, externalizing symptoms, neurocognitive measures, and ADHD related impairment while controlling for the influence of inattentive symptoms and biological sex. Internalizing, anxiety, and externalizing symptoms were measured by the Achenbach System of Empirically Based Assessments, Adult Self-Report (ASEBA). The ASEBA measure is similar to the TRF-ABPS in its use of self-report scales to examine symptoms indicative of internalizing, externalizing, or anxious symptomatology rooted in Achenbach's theory of TDFs. Reliability and validity of this measure has been established by its authors (Achenbach & Rescorla, 2003). Depressive symptoms were measured by the Center for Epidemiological Studies Depression Scale, a self-report measure examining symptoms associated with depression experienced within the past week (CES-D; Radloff, 1977). Participants in the moderate and severe SCT groups exhibited significantly higher scores on the ASEBA scale measuring anxiety and the CES-D measuring depression than those in the minimal SCT group (Kamradt et al., 2018). The neurocognitive findings in this study are detailed below.

In summary, the aforementioned studies have demonstrated that levels of SCT are positively related to internalizing symptoms such as anxiety and depression. Multiple studies have confirmed this relationship in children, older adolescents, and adults when controlling for symptoms of ADHD and demographic variables. The SCT subscale of the BAARS-IV demonstrated consistent and strong associations across studies with a variety of measures used to operationalize anxiety and depression. This pattern of similar findings across studies using a variety of measures is evidence of a consistent association

between SCT and symptoms of both anxiety and depressive symptoms. Furthermore, the results of these studies suggest that the relationship between ADHD and anxiety and depression may be partially explained by SCT (Becker, Langberg, et al., 2014). One theory on the close relationship between these constructs posits that SCT, anxiety, and depression may share several core features such as apathy, rumination/daydreaming, inactivity, and decreased effort (Smith & Langberg, 2017).

Neurological Research on ADHD

ADHD is a neurodevelopmental disorder characterized by difficulty sustaining attention, hyperactivity, and/or impulsive behavior. These symptoms are commonly attributed to structural differences in the brain and dysfunction in the creation and distribution of dopamine in the brain (Rubia et al., 2014; Wiers et al., 2018).

Brain Structure and Function in ADHD

The majority of brain imaging studies on ADHD has been conducted on children. These studies have used magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI) to examine abnormalities in the brains of those with ADHD. Structural anomalies in the frontal, parietal, striatal, and cerebellar regions of the brain, and the white matter networks that connect them are closely associated with ADHD symptomatologyin children (Rubia et al., 2014). The few published brain imaging studies of adults with ADHD have found moderate structural abnormalities in these same brain regions (Rubia et al., 2014). Meta-analyses of imaging studies have concluded that abnormalities in the basal ganglia are the most consistently found structural abnormality in the brains of those with ADHD (Rubia et al., 2014). The structures that make up the basal ganglia (including the caudate nucleus, putamen, globus pallidus, and substantia nigra) contribute to

processes such as voluntary motor movement and inhibitory control, the reward system and motivation, and dopamine synthesis (Kolb & Whishaw, 2015; Rubia et al., 2014).

Additional structural abnormalities include reduced grey matter volume in the prefrontal cortex of individuals with ADHD compared to typically developing individuals of the same age (Rubia et al., 2014). Rather than reflecting a consistent structural abnormality, longitudinal research has found that the difference is due to a delay in cortical development (Kolb & Whishaw, 2015; Shaw et al., 2007; Rubia et al., 2014). The human prefrontal cortex controls cognitive processes that allow the selection of behaviors or actions to engage in based on internal, external, and context cues. The prefrontal cortex also contributes to our knowledge of our autobiographical experiences and personal goals, which is referred to as autonoetic awareness (Kolb & Whishaw, 2015). Delayed development in the prefrontal cortex may contribute to the impaired self-regulation of behavior seen in ADHD due to impaired ability to attend to context cues and long-term goals.

Few studies have attempted to differentiate between subtypes of ADHD or between ADHD and SCT using brain-imaging techniques. One such study on ADHD compared the performance of school age children with ADHD-In to those with ADHD-C on a go/no-go task. Go/No-go tasks measure the ability to inhibit responding via button press to a decoy stimulus, which looks similar to a target stimulus to which the participant is supposed to respond to. During the go/no go task, functional and structural MRIs were taken. Analysis of performance revealed no significant differences between the ADHD-In and ADHD-C groups on performance of the go/no-go task itself. All participants exhibited activation of the temporoparietal junction and right ventrolateral

prefrontal cortex during successful inhibition on the go/no-go task. These areas have been found to be involved with detecting external cues for behaviors and signaling for the suppression of behavior, respectively (Solanto et al., 2009). Children with ADHD-In demonstrated a greater magnitude of activation in temporoparietal junction, despite the ADHD-In and ADHD-C groups having nonsignificant differences in performance. These findings imply that the brains of those with ADHD-In require greater activation of the temporoparietal junction to achieve the same level of performance as those with ADHD-C (Solanto et al., 2009).

A similar study compared fMRI scans of typically developing adolescents and adolescents with ADHD-In during a response inhibition task. The relationship between SCT symptomatology and levels of brain activation in different regions was examined using regression analysis. Activation in the superior parietal lobule during the inhibition task was negatively correlated with parent ratings of SCT symptoms (Fassbender et al., 2015). The superior parietal lobule receives sensory input from the eyes and hands, and assists the rest of the parietal lobe in integrating sensory information. The authors posit that the negative relationship between superior parietal lobule activation during the response inhibition task and SCT symptoms may represent impairment in the ability to reorient attention to a new stimulus (Fassbender et al., 2015). This theory is consistent with Posner's model of attention, which is discussed later.

The Dopaminergic System

Dopamine is an amine that functions as both a hormone and neurotransmitter in the human body. Nuclei in the substantia nigra and ventral tegmentum synthesize dopamine and distribute it via long axon pathways throughout the central nervous system.

Dopaminergic pathways consist of the nigrostriatal pathway and mesolimbic pathway (Kolb & Whishaw, 2015). The nigrostriatal pathway shuttles dopamine to the dorsal striatum, where it helps maintain normal motor control. The mesolimbic pathway carries dopamine to the nucleus accumbens and the prefrontal cortex. The nucleus accumbens is crucial to the reward system of the brain and motivation. The prefrontal cortex is responsible for complex cognitive functions such as planning, decision making, and social behavior. Decreases in dopamine availability in the mesolimbic pathway have been hypothesized to be the cause of deficits in decision-making, motivation, and reinforcement processes in ADHD (Sonuga-Barke, 2005; Volkow et al., 2009). Similar to studies showing reduced grey matter in adolescents with ADHD, slower development and pruning of neuronal pathways to the caudate nucleus (within the basal ganglia) was also observed in the brains of adolescents with ADHD. These differences disappeared or were negligible by the time these adolescents reached early adulthood (Rubia et al., 2014; Silk et al., 2009;).

The existing body of research into brain structure and function in ADHD is somewhat limited by small sample sizes, but interest continues to grow in imaging studies as a way to better understand the disorder (Rubia et al., 2014). ADHD is associated with structural and functional differences from typically developing brains in the frontal lobe, striatum, basal ganglia, and dopaminergic systems is associated with ADHD (Fassbender et al., 2015; Rubia et al., 2014; Shaw et al., 2007; Solanto et al., 2009; Sonuga-Barke, 2005; Volkow et al., 2009; Wiers et al., 2018).

Genetics of ADHD Genetic studies have found polymorphisms in genes responsible for encoding proteins of the dopamine transporter (DAT1) and the dopamine

receptors (D2 and D3) to be related to ADHD (Wiers et al., 2018). These proteins are responsible for dopamine reuptake from the synaptic cleft and uptake of dopamine into the postsynaptic neuron (Volkow et al., 2009). Stimulant medications prescribed for ADHD are thought to be effective because they work to increase the availability of dopamine in the synaptic cleft (Weirs et al., 2018). Studies have shown that higher levels of DAT1 available in the striatum are associated with better treatment response to stimulant medications in adults with ADHD (Krause, 2008).

The alleles associated with ADHD are commonly found in the general population and, therefore, lack the specificity necessary to represent a "biomarker" for ADHD. One line of research has explored epigenetic markers that could explain the interaction between genes and environment that impacts dopaminergic processes in ADHD. Epigenetic studies have found the methylation or expression of DAT1 gene in blood cells is associated with DAT availability in the striatum and substantia nigra as measured by positron emission tomography, and tissue and blood samples taken during postmortem autopsies of primate brains and the brains of humans with ADHD (Rajala et al., 2014; Weirs, 2018).

Brain changes in response to medication and psychotherapeutic treatment. Researchers use brain imaging techniques to examine changes in the structure of the brain as a way to measure treatment outcomes for psychotherapy and medications. These studies generally measure brain changes through prettreatment and posttreatment imaging or examining the relationship between pretreatment imaging and posttreatment measures of response to treatment. The efficacy of treatments for a wide variety of psychological disorders, including ADHD, has been investigated using these methods (Weingarten &

Strauman, 2015). Imaging studies of brains before and after receiving psychotherapeutic interventions or prolonged psychostimulant use have observed a normalizing effect, whereas the brains of those with ADHD become more similar to the brains of typically developing individuals.

For example, one study examined the efficacy of a "summer camp" style token economy program for children with ADHD. ADHD and control children without ADHD had functional MRI images taken during a go/no-go task before and after living and participating in the activities of the behavioral treatment program for ten days. Control children's inhibition greatly improved post treatment, but the ADHD children's performance did not significantly improve. The functional MRI images (fMRI) taken during the pretreatment administration of the go/no-go task revealed activation of the anterior cingulate cortex, right caudate nucleus, and dorsal lateral pre frontal cortex (DLPFC) bilaterally in both groups. However, activation in these areas pretreatment was higher in the controls compared to the ADHD group. Posttreatment, the between groups differences in fMRI brain activation in these regions were nonsignificant, with the ADHD children showing only slight, nonsignificant improvement in reaction time variability (a measure of basic attention) from pretreatment levels (Siniatchkin et al., 2012).

In another study, adults with ADHD who received 12 weekly sessions of CBT demonstrated improvements in brain connectivity and reduction in ADHD symptomatology. MRI imaging following treatment observed increased functional connectivity between the frontal-parietal network, the cerebellum and superior parietal lobule, as well as decreased self-reported ADHD symptoms. Increased bilateral

connectivity in the superior parietal lobule was significantly and negatively correlated with ADHD symptoms severity, indicating impaired connectivity in the superior parietal lobule may play a key role in ADHD and its amelioration (Wang et al., 2016).

Imaging studies examining the effect of psychostimulants on those with ADHD consistently find that stimulant use alters the structure and function of the brain. A literature review found 29 published imaging studies on changes to the structure and function of the brain in ADHD related to stimulant medication use. All studies examining structural differences between unmedicated participants with ADHD and control groups without ADHD found structural abnormalities in the brains of those with ADHD. Stimulant medication use was associated with attenuation of these structural abnormalities in many of the brain regions examined. The most consistent finding between these studies found stimulant use to treat ADHD was associated with greater volume of white matter in all lobes of the brain, and grey matter in the anterior cingulate cortex and splenium of the corpus callosum (Spencer et al., 2013). The majority of the functional imaging studies found that participants with ADHD demonstrate less activation in the striatum (including caudate and putamen), anterior cingulate cortex, and prefrontal cortex when subjected to tasks that require prolonged use of attention, executive functioning, or emotion regulation. These structural differences in the brains of those with ADHD were negligible from control groups following prolonged treatment with stimulant medications (Spencer et al., 2013).

Research into brain changes following interventions for ADHD demonstrated that those with ADHD exhibit less activation of the regions of the brain and neural networks involving attention, working memory, sensory integration and dopamine production. This

reduced activation can be ameliorated by evidence based-psychotherapy and treatment with stimulant medication, however these functional improvements do not always lead directly to improved ADHD symptomatology (Siniatchkin et al., 2012; Spencer et al., 2013; Wang et al., 2015).

Neuropsychological Functioning in SCT and ADHD

Although slow or inefficient cognitive abilities is considered one of the key features of SCT, research has produced mixed results when examining the relationship between SCT and various measures of cognitive abilities (Bauermeister et al., 2012; Becker & Barkley, 2018). Researchers have compartmentalized and operationalized cognitive abilities into a variety of domains such as executive functioning, working memory, and processing speed. Poor performance on measures of these constructs indicates that an individual's cognitive functions are notably slower or inefficient compared to the general population. This cognitive impairment is attributed to structural and functional differences in the brain that alters the way it functions compared to typically developing individuals.

Facets of cognitive functioning such as learning, working memory, processing speed, attention, visual-spatial skills, motor coordination, and verbal reasoning are believed to be localized to specific regions of the brain. Complex collaboration among these interdependent regions enables the brain to produce the sophisticated behaviors and thoughts that make up the human experience. Dysfunction or injury in one or more of these regions interrupts these interdependent processes and can influence or impair one's cognitive or behavioral functioning (Lezak et al., 2012). Thus, brain imaging technology can offer detailed images of alterations to the structure and function of the brain that

illuminate the neurological substrates of ADHD and explain some neuropsychological functioning.

Neuropsychological tests offer ways to measure broad concepts such as general intelligence or specific cognitive processes. One of the most widely accepted neuropsychological batteries is the Wechsler Adult Intelligence Scale - Fourth Edition (WAIS-IV). Completion of the standard test battery on the WAIS-IV produces a score of general intelligence and index scores representing the strength of processing speed, working memory, verbal ability, and perceptual reasoning compared to an age matched population sample.

A variety of neuropsychological measures exist that infer impairment in cognitive functioning through observation of an individual's performance on a task (Lezak et al., 2012). A meta-analysis of studies comparing participants with ADHD and control groups found performance on several neuropsychological measures to differ significantly. Specifically, the participants with ADHD consistently performed worse on the Trails A and Trails B test, which are neuropsychological measures of processing speed and executive functioning, respectively (Hervey et al., 2004; Reitan, 1993). Their performances on the digit span, digit symbol coding (Coding task), and arithmetic tests from the WAIS (measuring processing speed and working memory) were significantly worse with ADHD than the control groups' (Hervey et al., 2004; Wechsler, 2008;).

These neuropsychological measures of executive functioning, processing speed, and working memory held large to medium weighted effect sizes (Hervey et al., 2004). Whereas these particular measures held substantial effect sizes across studies, many other neuropsychological measures of working memory, processing speed, and executive

functioning had negligible findings across these studies. This pattern of inconsistent findings across neuropsychological measures that purportedly measure the same cognitive function was true for measures of attention, memory, and motor speed as well. These findings indicate that no single cognitive function is attributable to ADHD. Rather, those with ADHD exhibit widespread neuropsychological deficits and variability in multiple domains of cognitive functioning (Hervey et al., 2004).

SCT and Neuropsychological Functioning

There is a dearth of studies examining the relationships between SCT and measures of working memory and processing speed (Jacobson et al., 2018). One study examined children and adolescents with ADHD-IN and typically developing children and adolescents. Parents' ratings of their children's SCT symptoms and the participants' processing speed index (PSI) scores on the Wechsler Intelligence Scale for Children were used in this analysis. All participants' SCT ratings were factor analyzed, producing three factors consistent with the sleepy/sluggish, daydreamy, and low initiation symptom domains of SCT. The day dreamy and low initiation factors were significantly inversely associated with PSI scores with a small effect size, indicating SCT symptoms partially reflect "sluggishness" in their cognitive processes (Jacobson et al., 2018).

A study of SCT and ADHD in Puerto Rican children utilized mothers' and teachers' collateral report ratings of the participants' behavior and ADHD symptomatology and neuropsychological measures. The authors combined multiple collateral report and neuropsychological measures of behavior and cognitive functioning into composite scores that represented the participant's ability in different cognitive domains. These composite scores included measures of working memory, processing

speed, memory retrieval, interference control, and planning/problem solving. Separate confirmatory factor analyses were conducted for the teacher ratings and parent ratings of ADHD and SCT symptomatology to confirm the parent rating were accurately reflecting the inattentive, hyperactive/impulsive, and SCT domains of ADHD. Contrary to expectations, SCT was not associated with any of the collateral reports of cognitive functioning or executive functioning measures (Bauermeister et al., 2012). On the other hand, strong associations were found between ADHD-In symptoms and many of the cognitive functioning measures used. The authors acknowledged these relationships between inattention and cognitive functioning were not controlled for in the analyses, and may have overshadowed SCT's relationships with the cognitive functioning measures (Bauermeister et al., 2012).

Another cause for these disparate findings may be in the types of measures used to create the composite scores. The working memory composite was derived from neuropsychological measures of verbal working memory for numeric information, nonverbal working memory for spatial information, and nonverbal working memory for motor sequencing. In a similar way, the processing speed composite was derived from neuropsychological measures of rapid naming of stimuli, speeded motor, and nonlinguistic processing speed. It is possible SCT's lack of associations with the processing speed and working memory composites is due to the measures used to build these composites examining different loosely related functions rather than representing one uniform cognitive function (Bauermeister et al., 2012).

Kamradt et al. investigated the differences between adults with minimal, moderate, or severe levels of SCT in their anxiety, internalizing symptoms, and

externalizing symptoms. They also compared these groups on their performance on measures from a battery of neuropsychological tests. The three groups did not differ in their performance on measures of inhibition, interference control, vigilance, or sustained attention. Only those in the moderate SCT group exhibited significantly worse working memory (measured by performance on the WAIS-IV Digit Span task) compared to both the severe and mild SCT groups. This finding is unusual because it implies a parabolic relationship between SCT and working memory. One explanation for this finding put forth by the study's authors suggests that those with moderate SCT may be at particular risk for working memory impairment due to subtle diffuse neuropsychological impairment culminating to impair working memory (Kamradt et al., 2018).

Utility of Neuropsychological Testing for ADHD and SCT

The extant literature demonstrates inconsistency in the relationships between both ADHD and SCT and a variety of neuropsychological measures, especially those of working memory and processing speed. Although standardized neuropsychological measures are generally considered crucial to accurately measure cognitive functions, the validity of neuropsychological testing for diagnosing of ADHD has been called in to question. Prior research has found between 35% and 87% of those who meet *DSM* criteria for ADHD show no impairment on neuropsychological measures. As a result, the false negative rate of most neuropsychological measures is too high to be used as a diagnostic measure for ADHD (Barkley, 2019; Matier-Sharma et al., 1995; Ramsay, 2015). Clinicians advocating for best practices in the diagnosis of ADHD and SCT argue that clinical interviews and self-report measures of executive functioning are more ecologically valid tools for identifying the difficulties those with ADHD and SCT face

and have better predictive power (Barkley, 2011b, 2019; Barkley & Fischer, 2011; Pettersson et al., 2018). Although neuropsychological measures may not be the best way to diagnose ADHD in adults, these measures still provide valuable information on cognitive deficits, language issues, effort, and other potential issues that can affect learning and occupational functions and may, therefore, be more useful for identifying deficits and treatment planning, than diagnosis alone (Mapou, 2019).

Executive Functioning

Researchers examining the construct of executive functioning have defined it in different but closely related ways. Conceptualizations of executive functioning have attributed a variety of cognitive processes and abilities to executive functioning including goal-directedness, time management, hindsight, self-consciousness, inhibition, motivation, problem solving, interference control, and cognitive flexibility. As previously mentioned, executive functioning is a set of top-down mental abilities that requires the collaboration of different cognitive and neuropsychological processes. These processes work together to help an individual monitor and regulate behavior over time towards the achievement of a goal or to solve a novel problem. In other words, executive functioning determines how effective one is at accomplishing what they set out to do. (Barkley, 2011b; Diamond, 2013). Core aspects of executive functioning recognized by the National Institutes of Health are inhibition (self-control and resisting the urge to act impulsively), interference control (selective attention and cognitive inhibition), working memory, and cognitive flexibility (creative thinking and quickly and flexibly adapting to changing circumstances; Diamond, 2013).

Executive functioning deficits have been implicated in a variety of mental health disorders including addiction, conduct disorder, depression, obsessive compulsive disorder, schizophrenia, and ADHD. Executive functioning deficits have also been observed to be associated with social and physiological problems including obesity, school performance, vocational success, marital discord, and even criminal behavior across multiple studies and populations (Diamond, 2013). Different patterns of executive functioning have been found to differently associate with both ADHD and SCT in adults (Barkley, 2011b, 2011c, 2012; Willcutt et al., 2005).

The Barkley Deficits in Executive Functioning Scale (BDEFS) is a self- and other-report behavior rating scale developed to measure deficits in specific executive functioning domains and overall executive functioning in adults (Barkley, 2011b). These domains include self-management to time, self-organization/problem-solving, self-restraint, self-motivation, and self-regulation of emotion (Barkley, 2011b). The BDEFS domains are functionally different from neuropsychological measures of executive functioning because it assesses how dysfunction in executive functioning causes problems in everyday life over the past six months. Neuropsychological measures of executive functioning indicate how individuals perform on an executive functioning task in a controlled setting, typically for no longer than 30 minutes and may, thus, lack the external validity found in the BDEFS. Nonetheless, all BDEFS domains were largely found to be mildly to moderately correlated with several neuropsychological measures of cognitive functioning such as the Digit Span task from the WAIS-III, Conners' continuous performance task, and Stroop color word task (Barkley, 2011b; Smith et al., 2013).

The BDEFS domains were largely unrelated to measures of intelligence or academic achievement, with the exception of the deficits in self-organization/problem-solving domain holding a weak negative relationship with full scale IQ on the WAIS-IV (Barkley, 2011b). Preliminary research on the BDEFS found significantly more adults with ADHD rated themselves as having clinical levels of impairment in executive functioning than a clinical control group and a community control group (Barkley, 2011b). In another test comparing executive functioning tests and rating scales, the BDEFS and other tests of executive functioning were administered to adults with ADHD, adults who had ADHD in childhood that remitted in adulthood, and individuals who never exhibited symptoms of ADHD. Of these measures of executive functions, only the five subscales of the BDEFS were sensitive to differences between those with current ADHD from those whose symptoms remitted after childhood (Barkley, 2011b). These findings indicate the BDEFS holds good construct and discriminant validity, and is an ecologically valid measure of executive functioning deficits (Barkley 2011b, 2019).

Executive Functioning in ADHD and SCT

Barkley examined the differences in executive functioning between ADHD and SCT by comparing subscale scores on the BDEFS of groups of adults with ADHD only, SCT only, both ADHD and SCT, and a control group. The SCT-only group and the ADHD and SCT group both reported worse self-organization and problem solving abilities than the control or ADHD-only group. The combined ADHD and SCT group scored the lowest on the subscales of self-management to time, self-restraint, self-motivation, and self-regulation of emotion (Barkley, 2012). Stepwise regression analysis examined how much the participant's symptom ratings of SCT, ADHD-In, and ADHD-

H/I contributed to the variance in executive functioning subscale scores. SCT symptoms contributed the most variance in Self-Organization and Problem-Solving (54.5%), Self-Discipline (45.8%), and Self-Regulation of Emotion (44.5%), with ADHD-IN explaining less than 9% of the variance for each. ADHD-In symptoms contributed the most variance to Self-Management to (54.5%) and Self-Motivation (48.8%) with SCT symptoms explaining less than 5% of the variance for each (Barkley, 2012).

One study utilizing a nonclinical sample of college students explored the relationship between SCT scores on the BAARS-IV, executive functioning scores on the BDEFS, study skills, and general functional impairment. Path analyses were conducted to control for the influence of demographic information, anxiety, and depression, and ADHD symptoms on the relationships between SCT, executive functioning, study skills, and general functional impairment. SCT remained significantly associated with executive functioning deficits, poorer study skills, and general functional impairment. Two regression models examined the relative contribution of anxious, depressive, SCT, and ADHD symptoms as well as demographic information in predicting executive dysfunction. These analyses found that adding SCT to the model reduced the association between depressive symptoms and executive functioning to nonsignificance. Moreover, adding SCT to the model dramatically reduced the relationship between ADHD-In symptoms and executive functioning. In fact, SCT emerged as the strongest predictor of executive functioning deficits and added an additional 6% to the variance in executive functioning (Flannery et al., 2017).

The previously mentioned study by Leikauf and Solanto (2017) examined differences in relationships between SCT, ADHD symptoms, and a measure of executive

functioning in medicated and unmedicated adults with ADHD. They used self-report form of the BDEFS to measure executive functioning, self-report form of the BAARS-IV to measure SCT, the STAI and BDI-II to measure depression, and the Conners Adult ADHD Rating Scale-Self-Report (CAARS) to measure ADHD symptoms. In unmedicated participants, strong and significant correlations were observed between SCT scores and the BDEFS Summary score as well as between ADHD symptoms and the BDEFS Summary score. However, in the medicated group ADHD symptoms were not significantly correlated with the BDEFS Summary score. Only the self-restraint score was significant for those taking stimulant medications. SCT was a better predictor of overall executive functioning impairment, "above and beyond," ADHD or depression for both medicated and unmediated participants. These results seem to imply that, when ADHD symptomatology was reduced or managed by stimulant medication, the relationship between SCT and executive functioning persisted (Leikauf & Solanto, 2017).

In studies using the BDEFS to examine executive functioning's relationships with SCT and ADHD, SCT was found to be a significant predictor of deficits in all executive functioning domains on the BDEFS, even when controlling for confounding factors such as ADHD, internalizing symptoms, and demographic information (Jarrett et al., 2017; Leikauf & Solanto, 2017). These findings indicate that the BDEFS is a good measure of executive functioning impairment in those with both ADHD and SCT.

These studies demonstrate inconsistency and complexity in the relationships of ADHD and SCT to a variety of measures of neuropsychological and cognitive functioning, including measures of executive functioning, working memory, and processing speed. However, some measures of these constructs have demonstrated more

consistent relationships with SCT and ADHD than others. A meta-analysis of neuropsychological measures' relationships with ADHD by Hervey et al., found the subtests from the processing speed index and working memory index of the WAIS-IV consistently demonstrated significant large effect sizes in its associations with ADHD. The executive functioning domains measured by the BDEFS consistently demonstrated unique and independent relationships with ADHD and SCT symptomatology (Barkley, 2011b; Barkley, 2012; Flannery et al., 2017; Jarrett et al., 2017; Leikauf & Solanto 2017). These relationships are independent the association of SCT with classic ADHD symptomatology, and in some studies, SCT was a greater predictor of cognitive functioning than ADHD symptoms. According to Leikauf and Solanto (2017), "overall, these results demonstrate that SCT in adults is not exclusively a proxy for ADHD symptom severity or internalizing symptomatology" (p. 709), but rather represents its own syndrome that predict deficits in cognitive functioning.

Summary of Literature Review

SCT and ADHD are two highly related disorders that co-occur in 46% of all cases of ADHD and 54% of all cases of SCT (Barkley, 2012). SCT symptomatology shares many features with the inattentive subtype of ADHD, but extensive testing has demonstrated that these disorders are distinct from one another (Barkley, 2011a, 2012; Becker et al., 2016; Becker & Barkley, 2018; Becker, Marshall, & McBurnett, 2014; Garner et al., 2010; Wåhlstedt & Bohlin, 2010).

SCT scores are, independently of ADHD symptoms, associated with impairment in social, academic, and emotional functioning (Becker & Barkley, 2018). SCT was also associated with internalizing symptoms, such as depression and anxiety, when controlling

for ADHD symptomatology (Bauermeister et al., 2012; Becker, Langberg, et al., 2014; Carlson & Mann, 2002; Kamradt et al., 2018). In some instances, SCT held stronger relationships with internalizing symptomatologythan ADHD (Becker, Langberg, et al., 2014). This relationship between SCT and internalizing disorders may be due to shared features of these disorders such as apathy, rumination/daydreaming, inactivity, and decreased effort (Smith & Langberg, 2017). High levels of effortful control and low stress levels are protective factors against internalizing disorders (Gulley, et al., 2017).

Both ADHD and SCT have been shown to be associated with impairment in a variety of neuropsychological and cognitive domains, such as executive functioning, working memory, processing speed, attention, and memory. However, these relationships are inconsistent and vary in significance and effect size from study to study (Bauermeister et al., 2012; Hervey et al., 2004; Jacobson et al., 2018; Kamradt et al., 2018). This is possibly due to the limited utility of neuropsychological measures in these populations and diffuse deficits across multiple neuropsychological and cognitive domains in both ADHD and SCT (Barkley, 2019; Hervey et al., 2004; Mapou, 2019).

Hypothetical Models of SCT

The process model of depletion argues that an individual's self-control wanes as their motivation and attention shifts away from goal directed behavior (Inzlicht & Schmeichel, 2012). External manipulations of motivation have been shown to reduce the impairment in basic attention in those with ADHD, as posited by this model (Dekkers et al., 2017; Inzlicht & Schmeichel, 2012). Second, conceptualization of ADHD and SCT in Posner's model of attention argues that SCT is characterized by deficits in the orienting

stage of attention, whereas ADHD is the product of deficits in the alerting, orienting, and executive functioning aspects of attention (Barkley, 2016; Becker & Willcutt, 2018).

In light of these two theories and research on the relationships between ADHD and SCT, SCT symptomatology may be the product of interactions between internalizing symptomatology and impaired cognitive functioning. Consider the case of a hypothetical individual who experiences impairment in working memory and processing speed, with comorbid internalizing symptoms. When a situation or task that requires mental effort arises, their impaired cognitive functions are already taxed by internalizing symptomatology, reducing their capacity to orient to the new situation and decreasing their motivation to expend further effort to engage with the new situation or fully exercise their executive functions. Internalizing symptoms such as anxiety, depression, somatic complaints, and traumatic stress may lead to rumination (daydreaming about negative emotional experiences) and require sustained effort to cope with the negative aspects of these symptoms. These consequences are reflected in the day dreamy and low initiation aspects of SCT (Jacobson et al., 2018). Following this logic, SCT may be better conceptualized as a transdiagnostic factor that is present in individuals with neuropsychological impairments or a separate neurodevelopmental disorder in its own right.

SCT could also be conceptualized as a syndrome whose symptoms reflect deficits in executive functions and the internalization of these challenges. The theory of TDFs posits that underlying mental health problems express themselves as different mental health disorders with different symptoms according to an individual’s personal

characteristics and life experiences. A commonly accepted TDF paradigm is the internalization or externalization of negative affect (Krueger & Eaton, 2015).

Multiple researchers have proposed that deficits in a specific executive function or broad deficits in executive functioning are the source of the inattentive, hyperactive, and impulsive symptoms of ADHD (Willcutt et al., 2005). A study of SCT and ADHD found SCT contributed the majority of the variance to the Self-Organization and Problem-Solving, Self-Discipline, and Self-Regulation of Emotion subscales of the BDEFS , whereas inattentive symptoms of ADHD explained less than 9% of the variance for each. Inattentive symptoms explained the majority of the variance for the remaining Self-Management and Self-Motivation subscales, whereas SCT explained less than 5% of the variance for each (Barkley, 2012). Perhaps deficits in these or other specific executive functions are also the source of the symptoms of SCT.

Considering SCT and executive dysfunction in the context of internalization of mental health challenges, it is theoretically possible that SCT represents a novel internalizing expression of executive dysfunction. The hyperactive and impulsive symptoms of ADHD may reflect an externalization of executive dysfunction or inhibition, whereas the inattentive symptoms could reflect an internalization of executive dysfunction or inhibition. If an individual is experiencing executive dysfunction in organization, self-discipline, or emotion regulation and they have a tendency to internalize their problems, their unique blend of personal characteristics and life experiences may result in SCT symptomatology. This could also partially explain SCT's strong relationships with internalizing symptoms. An internalizing disorder such as depression reflects the internalization of negative affect. In the same way, SCT may

reflect internalization of executive dysfunction. An individual who has a tendency to internalize their problems would be more likely to develop an internalizing mood disorder, such as depression, and internalize their executive dysfunction, which could be expressed as SCT.

Chapter 3: Method

The study examined the relationships between SCT, ADHD, cognitive functioning, and internalizing symptoms in adults diagnosed with ADHD, employing a combined cross-sectional between subjects design and correlational/regression design using existing data.

Participants

This study utilized existing data collected from 143 adult subjects who have previously received a psychological assessment at a university-based, adult ADHD outpatient specialty clinic in a large urban area located in the Northeastern United States.

Inclusion and Exclusion Criteria

This study examined existing data. Subjects were those 18 years old or older assessed at intake by clinician rating to meet the full symptom criteria for the Inattentive, Hyperactive/Impulsive, or Combined subtype of ADHD, as well as those who meet criteria for SCT. ADHD criteria were established by the Conners Adult ADHD Rating Scale - Self-Report (CAARS), and clinician ratings on the Structured Clinical Interview for DSM-5 Disorders (SCID-5). SCT criteria were established by the SCT subscale of the BAARS-IV.

Individuals diagnosed with severe psychiatric issues, such as a co-occurring psychotic disorder and severe current substance use disorders, as established by the SCID-5 administered as part of the psychological assessment, clinician rating, or previous diagnosis, were excluded from the study. Of the 143 subjects, only three met criteria for moderate substance use disorders. Substance use disorders have been identified as commonly occurring in individuals with ADHD (Zulauf, Sprich, Safren, &

Wilens, 2014). These individuals were retained for the analyses because vulnerability to substance use disorders is an important part of the ADHD profile.

Measures of ADHD and SCT Symptoms

Barkley Adult ADHD Rating Scales-Fourth Edition (BAARS-IV)

The BAARS-IV is a 27 item scale, available in self- and other-report rating versions, designed to measure the symptoms of ADHD (Barkley, 2011a). The items are organized into four subscales: three that correspond with the *DSM-IV* inattentive, impulsive, and hyperactive symptoms of ADHD, and a fourth that measures the symptoms of SCT. Scores on the inattentive and SCT subscales can range from 9 to 36, with higher scores indicating greater levels of symptomatology. Scores on the hyperactive subscale range from 5 to 20. Scores on the impulsive subscale range from 4 to 16. Higher scores on a subscale of the BAARS-IV reflect a greater number and/or magnitude of endorsed symptoms of ADHD or SCT. Participants and an informant of their choice are asked to rate the frequency with which the participant engages in or experiences each of the symptoms of ADHD or SCT from 1 (*never or rarely*) to 4 (*very often*).

Subjects' responses on the four BAARS-IV subscales are summed to create a total raw score for each subscale. The raw score is used to calculate a statistic reflecting each subject's percentile rank compared to others in the same age group for each symptom domain. A subject's percentile score represents the portion of individuals in the normative sample the individual scored higher than on the subscale. Scores in the 93rd percentile or higher reach clinically significant levels of the symptom domain and indicate the presence of ADHD or SCT (Barkley, 2011a).

The BAARS-IV subscales were used to operationalize ADHD and SCT in two ways. Analyses examining ADHD and SCT symptoms from a continuous perspective used subjects' percentile ranks for each symptom domain. Analyses that conceptualized ADHD symptom domains and SCT from a categorical perspective will use the 93rd percentile score on each symptom domain as a cutoff score for identifying clinically notable levels of the symptom domain (Barkley, 2011a, 2012). The BAARS-IV subscales were found to have satisfactory internal consistency (Cronbach's α ranged from 0.776 to 0.914) and test-retest reliability (0.66-0.76; Barkley, 2011a). Further testing of the BAARS-IV using Widaman's (1985) method supported convergent validity with a *DSM-IV* checklist of childhood ADHD (X^2 = 903.0, $p < 0.001$) and discriminant validity between self and informant forms of the BAARS-IV (X^2= 1166.9, $p = 0.01$) using multitrait multimethod approach (Leopold et al., 2015).

Conners Adult ADHD Rating Scale - Self-Report (CAARS)

The CAARS is a 66-item self-report measure designed to examine the symptoms of ADHD in adults. Participants respond to each item by rating themselves from 0 (*not at all*, *never*) to 3 (*very much*, *very frequently*) based on their level of agreement with each item or their reported frequency of the behavior described by each item. The items on the CAARS contribute to four scales, that measure common symptoms of ADHD, as well as three scales that reflect the *DSM-IV* symptom criteria for ADHD. The *DSM-IV* Inattentive Symptoms subscale and *DSM-IV* Hyperactive/Impulsive Symptoms subscale were used as continuous measures to operationalize the symptoms of ADHD. This allowed for the severity of the subjects' inattentive and hyperactive/impulsive symptomatology to be measured continuously rather than merely identifying whether or

not they meet criteria for an ADHD diagnosis. Scores on the *DSM-IV* Inattentive Symptoms subscale and IV Hyperactive-Impulsive Symptoms subscale range from 0 to 27, with higher scores indicating higher levels of ADHD symptomatology. The raw subscale scores are converted to T scores, with a T score of 65 or greater indicating clinically notable levels of the symptoms domain. The *DSM-IV* Inattentive Symptoms subscale and IV Hyperactive-Impulsive Symptoms subscale of the CAARS have been found to have excellent discriminant validity; comparison study of adults who met *DSM* criteria for ADHD and an age- and gender-matched control group who did not meet *DSM* criteria for ADHD found these subscales had an 85% correct classification rate (Conners et al., 1999).

Measures of Internalizing Symptoms

Internalizing symptoms are operationally defined as scores on the BDI-II, Penn State Worry Questionnaire (PSWQ), the Affect subscale of the BADDS, and Neuroticism scale of the NEO-PI-R. In several studies, these measures of internalizing symptoms have demonstrated good convergent validity, with significant moderate relationships among them. In a prior study using data from the same university-based adult ADHD outpatient specialty clinic, the Neuroticism scale of the NEO-PI-R had good convergent validity with the PSWQ ($R^2 = 0.294$, $p < 0.08$) and the BDI-II ($R^2 = 0.343$, $p < 0.08$; Serine, 2016). The Neuroticism scale has also been found to be associated with difficulty regulating emotions as measured by the Affect scale of the BADDS ($R^2 = 0.388$, $p < 0.001$; Di Nicola et al., 2014). In a study of internalizing symptoms a sample of Caucasian and African American adults, scores from the BDI-II were significantly related

to scores on the PSWQ ($R^2 = 0.30$, $p < 0.01$; Chapman, Kertz, & Woodruff-Borden, 2009).

Brown Attention Deficit Disorder Scale - Adult Version (BADDS)

The BADDS is a 40-item self-report scale designed to screen for common difficulties and core features of the Inattentive symptoms of ADHD, based on the executive dysfunction model of ADHD (Brown, 1996; Brown & Whiteside, 2003). The BADDS is unique among measures of adult ADHD in that it identifies both ADHD symptomatology and signs of executive dysfunction that those with ADHD sometimes face. Each item on the BADDS describes a problem or symptom associated with ADHD. Participants rate how much each item has been a problem for them in the past 6 months from 0 (*never*) to 3 (*almost every day*). Each item on the BADDS contributes to a total score as well as one of five subscale scores, which measure difficulty (a) activating or organizing for work, (b) sustaining attention , (c) sustaining mental effort on boring or difficult tasks, (d) managing affect and emotion that could interfere with work, and (e) problems with working memory or recall. Only the affect subscale of the BADDS (BADDS-Affect) was used in these analyses as a measure of participants' capacity to regulate their internalizing symptoms in the analyses. Raw scores on the BADDS-Affect can range from 0 to 21. This score is converted to a T score, with T scores of 65 or greater indicating clinically notable levels of difficulty managing affect and emotion The BADDS has demonstrated satisfactory test-retest reliability ($r = 0.87$) and excellent internal consistency (Cronbach's $\alpha = 0.96$; Brown & Whiteside, 2003). The Affect subscale has demonstrated good convergent validity with the Internalizing problems subscale of the Behavior Assessment System for Children ($R^2 = 0.52$), and the Emotional

Control subscale of the Behavior Rating Scale of Executive Function (R^2 = 0.66; Brown, 1996).

Penn State Worry Questionnaire (PSWQ)

The PSWQ is a self-report measure designed to measure the severity of worry (Meyer et al., 1990). Participants respond to a series of 16 items on their tendency to worry, rating the statements from 1 (not at all typical of me) to 5 (very typical of me). Five of the items are reverse scored. Scores on the PSWQ range from 16 to 80. Higher total scores represent greater frequency and severity of worry. The PSWQ was used to measure the anxiety and worry component of internalizing symptoms in the planned analyses. Individuals with ADHD may underreport their level of impairment on self-report measures due to a lack of self-awareness on their behavior (Manor et al., 2012). By focusing on worry rather than other behavioral features of anxiety, the PSWQ has higher construct validity for those with ADHD than other measures of anxiety (Meyer et al., 1990). The PSWQ has demonstrated good internal consistency (Cronbach's α = .83-.93) and test-retest reliability (r = .74-.93) (Brown et al., 1992; Molina & Borkovec, 1994). The PSWQ has demonstrated good discriminant validity and captures features of generalized anxiety such as the apprehensive and uneasy symptoms of anxiety often expressed by those with co-occurring anxiety and ADHD (Meyer et al., 1990; Ramsay, 2015).

Beck Depression Inventory, Second Edition (BDI-II)

The BDI-II is a 21-item self-report measure designed to assess severity of depression (Beck et al., 1996). Participants rate the severity of their depression by endorsing different statements grouped by key symptoms of depression such as sadness,

self-dislike, social withdraw, loss of appetite, irritability, feelings of guilt, suicidality, and anhedonia. These statements are rated from 0 to 3 and summed to create a total score. Scores on the BDI-II range from 0 to 63. Higher scores on the BDI-II represent more severe symptoms of depression. The BDI-II has demonstrated high internal consistency (Cronbach's $\alpha = .91$) and test-retest reliability ($r = .93$) (Beck et al., 1996). Further testing of the BDI-II found it demonstrates moderate to high convergent validity with the Short Form General Health Survey ($R^2 = 0.42$, $p < 0.01$; Arnau et al., 2001).

Revised NEO Personality Inventory (NEO-PI-R)

The NEO-PI-R is a 240-item measure of the five personality domains in the five factor model of personality: Neuroticism, Openness, Extraversion, Agreeableness, and Conscientiousness. Items on the NEO-PI-R consist of statements about oneself that are rated on a Likert scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*). These items each contribute to subscale score reflecting one of the five personality domains. Scores on each subscale range from 0 to 192. These raw scores are converted to standardized T scores. T scores greater than 65 are in the very high range. T scores between 55 and 65 are in the high range. T scores between 45 and 55 are in the average range. T scores between 35 and 45 are in the low range. T scores below 35 are in the very low range (Costa & McCrae, 1992; Serine 2016). Internalizing behavior was assessed using the personality factor of neuroticismbecause it reflects symptoms of anxiety and depression as well as personality traits related to internalizing symptoms, such as self-consciousness and feelings of vulnerability.

Research on use of the NEO-PI-R in those with ADHD has found high neuroticism, low agreeableness, and low conscientiousness are common personality traits

shared among this population (Parker et al., 2004). The chronic restlessness and higher levels of physiological arousal characteristic of hyperactive symptoms of ADHD can be mistaken for anxiety by some measures (Meyer et al., 1990; Ramsay, 2015). The NEO-PI-R measures stable personality traits and the cognitive aspects of anxiety and other internalizing symptoms (Costa & McCrae, 1992). This allows it to measure internalizing symptomatology, without being overly influenced by confounding physiological symptoms of hyperactivity. Multiple studies have demonstrated that the personality domains of the NEO-PI-R hold excellent convergent validity with other well established measures of similar personality traits. This includes the relationship between NEO-PI-R Extraversion and the Extroversion scale of the Myers Briggs Type Indicator (R^2 = 0.55), NEO-PI-R Neuroticism and the Succorance scale of the Adjective Checklist (R^2 = 0.36), and NEO-PI-R Conscientiousness and the Endurance scale of the Adjective checklist (R^2 = 0.28; Costa & McCrae, 1992). The NEO-PI-R has also demonstrated good test-retest reliability (r = 0.52 to 0.81) and interrater reliability (r = 0.86 to 0.95) across multiple studies (Costa & McCrae, 1992).

Measures of Cognitive Functioning

Cognitive functioning was operationally defined as subjects' performance on the Coding task and Digit Span task of the WAIS-IV, and their Total Executive Functioning summary score of the BDEFS. These measures of cognitive functioning have demonstrated good convergent validity with one another, with small to moderate significant correlations ranging from .15 to .52 (Barkley 2011b; Wechsler, 2008).

Barkley Deficits in Executive Functioning Scale (BDEFS)

The BDEFS is a behavior rating scale developed to measure deficits in executive functioning in adults. The 89 items each contribute to five subscale scores on the BDEFS. Each subscale represents a different aspect of executive functioning that contributes to impairment in daily life activities. There is also a BDEFS Total Executive Functioning Summary score (BDEFS Summary score). These subscales are (a) Self-Management to Time, (b) Self-Organization/Problem Solving, (c) Self-Restraint, (d) Self-Motivation, (e) and Self-Regulation of Emotion as well as a BDEFS Summary score. The latter is calculated by summing the responses to all of the items on the BDEFS. Higher scores on a subscale of the BDEFS reflect greater impairment of executive functioning. Scores on the Self-Management to Time subscale range from 21 to 84. Scores on the Self-Organization/Problem Solving subscale range from 24 to 96. Scores on the Self-Restraint subscale range from 19 to 76. Scores on the Self-Motivation subscale range from 12 to 48. Scores on the Self-Regulation of Emotion subscale range from 13 to 52. The BDEFS Summary score can range from 89-356. Higher scores reflect greater dysfunction in the executive functioning domain, whereas lower scores reflect intact executive functioning.

The BDEFS Summary score is calculated by summing all items. This is converted to a percentile rank based on a subject's gender and age group. A subject's percentile score represents the portion of individuals in the normative sample they scored higher than on the BDEFS. An ADHD-EF index score is calculated from the sum of 11 items distributed throughout the measure that represent common executive functioning difficulties individuals with ADHD experience. The subscale scores, total executive functioning summary score, and ADHD-EF index score all demonstrated satisfactory

internal consistency (Cronbach's α ranged from 0.958 to 0.842) and test-retest reliability (*r* ranged from 0.62 to 0.90; Barkley, 2011b). Research validating the BDEFS measure found it has good convergent validity with the Coding task from the WAIS-IV (R^2 = 0.27; Barkley, 2011b). Research examining the BDEFS and a measure of goal-directed selective attention in a sample of college students with ADHD found support for the discriminant validity of the BDEFS (Dehili et al., 2017).

Wechsler Adult Intelligence Scale-Fourth Edition (WAIS-IV)

The WAIS-IV is an intelligence test battery designed to measure intellectual functioning in specific cognitive domains and general intellectual ability. Subjects were administered select subtests from the WAIS-IV as part of the ADHD evaluation process. This study used the Coding task and the Digit Span task as measures of impaired cognitive processes. The Coding task is a test of cognitive processing speed. The Digit Span task measures auditory working memory for numbers. Extensive testing has proven the reliability and validity of the WAIS-IV and its subtests in a variety of populations (Wechsler, 2008).

Procedures

This was a secondary analysis of data were gathered from new clients during intake at a university-based, adult ADHD outpatient specialty clinic in a large urban area. The aforementioned measures were completed as part of a comprehensive ADHD assessment. Research assistants and the administrators of the psychological assessment recorded the data from these measures, de-identified it, and copied the data into an SPSS data file. Human subjects research utilizing this data set was approved by a Institutional Review Board (IRB). Subjects had already completed the evaluation and signed a consent

form agreeing to let their private information be used in research studies and informing them of the minimal risk to the participants. This study utilized the data of participants who met the requirements of the inclusion and exclusion criteria collected between January 2014 and May 2019 for the analyses.

Chapter 4: Results

Diagnostic Group Creation

This study examined the relationship between ADHD, SCT, internalizing symptoms, and cognitive functioning. The sample consisted of 143 participants who presented for a comprehensive evaluation at an adult ADHD outpatient specialty clinic. Data were analyzed across the following four diagnostic subgroups: ADHD + SCT, ADHD + subclinical SCT, ADHD only, and SCT only; they could be distinguished from one another based on the degree of self-reported internalizing symptoms and impaired cognitive functioning. The study also examined which co-occurring internalizing symptoms and aspects of cognitive functioning were most predictive of SCT.

These four groups were created by sorting the participants according to their score on the SCT subscale of the BAARS-IV. Cutoff percentile scores for each group were determined by following Barkley's validation research on the BAARS-IV, which found the SCT subscale scores at the 93rd percentile or higher reflected clinically significant levels of SCT symptomatology (2011a). Those whose SCT scores were in the Borderline range as outlined in the BAARS-IV manual (85th to 92nd percentile) do not meet criteria for SCT, while still having notable levels SCT symptomatology. This range of scores reflects subclinical levels of SCT (Barkley, 2011a).

The ADHD + SCT group consisted of participants who met or exceeded the 93rd percentile for their reference age group on the SCT subscale (the threshold for clinically significant levels of SCT outlined in the BAARS-IV manual) and met criteria for an ADHD diagnosis, as determined by the CAARS and SCID-5, which was part of the original comprehensive evaluation. Seventy-four subjects from the data set met criteria

for ADHD + SCT and had all relevant data needed to conduct the analyses. The ADHD + subclinical SCT group consisted of 36 subjects whose SCT subscale scores were between the 85th and 92nd percentile for their reference age group, received an ADHD diagnosis, and had all relevant data needed to conduct the analyses.

The ADHD-only group consisted of 26 subjects who received an ADHD diagnosis, their SCT subscale score was below or equal to the 84th percentile for their reference age group, and had all relevant data needed to conduct the analyses. The SCT-only group consisted of seven subjects who met or exceeded the 93rd percentile for their reference age group on the SCT subscale, had all relevant data needed to conduct the analyses, and either did not receive an ADHD diagnosis or received a diagnosis of Other Specified ADHD. This diagnosis is used by the adult ADHD outpatient specialty clinic to communicate that an individual has clinical levels of SCT symptomatology, but does not meet full criteria for an ADHD diagnosis. Means and standard deviations for each diagnostic group's scores on the measures of internalizing symptoms and cognitive functioning are presented in Table 1.

Demographic Analysis

The sample consisted of 93 males and 50 females. The mean age was 34.17, with a standard deviation of 12.69, a median age of 31, and an age range of 18 to 72. Subjects identified as 77.6% Caucasian, followed by 10.5% other, 3.5% African American, 3.5% Hispanic, and 2.8% Asian. Data on ethnicity was not available for three subjects. Subjects had a mean education level of 15.74 years, with a standard deviation of 2.21, and range of 11 to 21.

Table 1

Internalizing Symptoms and Cognitive Functioning Across the Diagnostic Groups

Diagnostic Group		BDI-II	PSWQ	BDEFS Summary	BADDS-Affect	Coding Task	Digit Span	NEO-Neuroticism
ADHD + SCT (n = 74)	*M*	18.88	57.95	96.00	67.50	9.41	10.69	59.01
	SD	11.08	14.17	4.93	12.58	2.86	3.03	12.58
ADHD + subclinical SCT (n = 36)	*M*	14.64	50.06	90.47	59.39	9.81	10.19	56.72
	SD	8.69	14.43	12.28	12.02	2.66	2.66	10.23
ADHD-only (n = 26)	*M*	10.92	48.46	88.58	60.58	10.35	10.19	55.50
	SD	7.41	16.36	8.87	10.91	3.25	1.96	11.97
SCT-only (n = 7)	*M*	22.71	45.57	94.57	68.43	9.43	11.57	59.71
	SD	19.63	24.67	6.43	16.07	4.28	2.70	18.42
Total sample (N = 143)	*M*	16.55	53.63	93.19	64.24	9.68	10.52	57.83
	SD	10.91	15.76	8.67	12.79	2.95	2.75	12.21

Note: BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; BDEFS Summary = Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary % score; BADDS-Affect = Brown ADD Scales-Affect, T Score; Coding Task = WAIS-IV Digit Symbol Coding Scaled Score; Digit Span task = WAIS-IV Digit Span, Scaled Score; NEO-Neuroticism = NEO-Personality Inventory-Neuroticism-T Score.

Hypothesis 1 Analysis

It was hypothesized that there would be a significant difference between subjects diagnosed with ADHD + SCT, ADHD + subclinical SCT, ADHD only, and SCT only, with those diagnosed with ADHD + SCT having higher levels of internalizing symptoms than the other groups. Internalizing symptoms were operationally defined as scores on the BDI-II, Penn State Worry Questionnaire (PSWQ), the affect subscale of the Brown Attention Deficit Disorder Scale-Adult Version (BADDS), and Neuroticism scale of the NEO-PI-R.

A multiple analysis of variance (MANOVA) was conducted to examine whether subjects' scores on the BDI-II, PSWQ, BADDS-Affect, and NEO-Neuroticism differed significantly between those who exhibited clinical levels of ADHD + SCT, ADHD + subclinical SCT, only SCT, and only ADHD. Diagnostic category served as the independent variable for this study.

The assumptions of the MANOVA were tested by examining the normality, linearity, homoscedasticity, and multicollinearity of the data. To test the normality of the data, the skew and kurtosis of the data were calculated for the relevant variables. The skew and kurtosis of the variables used in these analyses were found to be within normal limits. Levene's test was used to examine the homogeneity of the variance across the groups. The results of Levene's test (Table 2) found the variance was unequal between the four diagnostic groups for subjects' scores on the BDI-II, $F(3, 139) = 5.409$, $p = 0.002$. The variance was also unequal for subjects' scores on the PSWQ for the four diagnostic groups, $F(3, 139) = 3.345$, $p = 0.021$.

The variance-covariance ratio was tested using Box's test of equality of covariance matrices. The Box's M value of 55.468 was found to be not significant (p = 0.02), indicating the assumptions of homogeneity covariance were met between the diagnostic groups. Due to unequal sample size, violation of the assumptions of equal variance, and groups differing along more than one variate, Pillai's trace was used as the significance statistic because it is more robust than the other statistics to violations of model assumptions (Field, 2013; Olson, 1974).

Table 2

Levene's Test of Equality of Error Variances in Measures of Internalizing Symptoms

	F	*df*1	*df*2	*p*
NEO-Personality Inventory-Neuroticism	2.382	3	139	.072
Beck Depression Inventory-II	5.409	3	139	.002
Penn State Worry Questionnaire	3.345	3	139	.021
Brown ADD Scales-Affect, T Score	1.512	3	139	.214

[a]Design: Intercept + Diagnostic group

Scores from the BADDS-Affect, BDI-II, PSWQ, and Neuroticism scale of the NEO-PI-R were tested for linearity and multicollinearity. As required for MANOVA, these dependent variables demonstrated significant correlations with one another, ranging from 0.244 to 0.517 ($p \leq 0.01$). These correlations are provided in Table 3. Bonferroni corrections were used to account for the potential of increased Type I experiment-wise

error rate caused by conducting multiple statistical tests in this study. To correct for the experiment-wise error rate of three comparisons the critical *p* value was set at 0.01, with a desired power level of 0.80. Using Pillai's trace, there was a significant effect of diagnostic group on subjects' scores on the measures of internalizing symptoms, $V = 0.198$, $F(12, 414) = 2.439$, $p = .004$. The observed power for this MANOVA was .897. Results from the multivariate tests are provided in Table 4.

Table 3

Summary of Pearson Correlations Between Measures of Internalizing Symptoms

	NEO- Neuroticism	BDI-II	PSWQ	BADDS-Affect
NEO-Personality Inventory-Neuroticism, T Score	-	.244**	.409***	.328***
Beck Depression Inventory-II	.244**	-	.255**	. 517***
Penn State Worry Questionnaire	.409***	.255**	-	.447***
Brown ADD Scales-Affect, T Score	.328***	.517***	.447***	-

Note: BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; BADDS-Affect = Brown ADD Scales-Affect, T Score; NEO-Neuroticism = NEO-Personality Inventory-Neuroticism-T Score.

**$p < .01$ one-tailed, ***$p < .001$ one-tailed

Table 4

Multivariate Tests for Differences in Internalizing Symptoms Between Groups[a]

Intercept	Value	*F*	Hyp. *df*	Error *df*	*p*	Partial Eta Squared	Noncent. Parameter	Observed Power[d]
Pillai's Trace	.953	691.63[b]	4	13	.000	.953	2766.53	1.00
Wilks' Lambda	.047	691.63[b]	4	136	.000	.953	2766.53	1.00
Hotelling's Trace	20.342	691.63[b]	4	136	.000	.953	2766.53	1.00
Roy's Largest Root	20.342	691.63[b]	4	136	.000	.953	2766.53	1.00
Diagnostic group								
Pillai's Trace	.198	2.44	12	414	.004	.066	29.26	.897
Wilks' Lambda	.812	2.46	12	360.11	.004	.067	25.90	.943
Hotelling's Trace	.220	2.46	12	404	.004	.068	29.57	.971
Roy's Largest Root	.146	5.026[c]	4	138	.001	.127	20.11	.959

Note. Hyp. *df* = Hypothesis degrees of freedom

[a]Design: Intercept + Diagnostic group

[b]Exact statistic

[c]The statistic is an upper bound on *F* that yields a lower bound on the significance level.

[d]Computed using $\alpha = .01$

A univariate ANOVA revealed subjects' NEO-Neuroticism scores did not differ significantly between the diagnostic groups, $F(3, 139) = 0.698$, $p = 0.555$. Separate univariate ANOVAs also revealed significant effects of diagnostic group on the BDI-II, $F(3, 139) = 4.914$, $p = 0.003$; PSWQ, $F(3, 139) = 4.286$, $p = 0.006$; and BADDS-Affect, $F(3, 139) = 4.617$, $p = 0.004$. The observed power for these univariate ANOVAs ranged from 0.068 to 0.751. The results of these univariate ANOVAs are provided in Table 5.

Table 5

Univariate Analysis of Variance Tests Comparing Between-Groups Effects for Internalizing Symptoms

Dependent Variable		Type III *SS*	*df*	*MS*	*F*	*p*	Observed Power
Corrected model	NEO-Neuroticism	313.835[a]	3	104.61	.698	.555	.068
	BDI-II	1621.871[b]	3	540.62	4.914**	.003	.751
	PSWQ	2987.508[c]	3	995.84	4.286**	.006	.670
	BADDS-Affect	2105.318[d]	3	701.77	4.617**	.004	.715

	Dependent Variable	Type III *SS*	*df*	*MS*	*F*	*p*	Observed Power
Intercept	NEO-Neuroticism	239602.529	1	239602.53	1598.260***	.000	1.000
	BDI-II	20258.501	1	20258.50	184.126***	.000	1.000
	PSWQ	183360.727	1	183360.73	789.177***	.000	1.000
	BADDS-Affect	294155.445	1	294155.45	1935.314***	.000	1.000
Diagnostic group	NEO-Neuroticism	313.835	3	104.61	.698	.555	.068
	BDI-II	1621.871	3	540.62	4.914***	.003	.751
	PSWQ	2987.508	3	995.84	4.286***	.006	.670
	BADDS-Affect	2105.318	3	701.77	4.617***	.004	.715
Error	NEO-Neuroticism	20838.137	139	149.92			
	BDI-II	15293.486	139	110.03			
	PSWQ	32295.848	139	232.34			
	BADDS-Affect	21127.116	139	151.99			

	Dependent Variable	Type III *SS*	*df*	*MS*	*F*	*p*	Observed Power
Total	NEO-Neuroticism	499424	143				
	BDI-II	56095	143				
	PSWQ	446567	143				
	BADDS-Affect	613449	143				
Corrected total	NEO-Neuroticism	21151.972	142				
	BDI-II	16915.357	142				
	PSWQ	35283.357	142				
	BADDS-Affect	23232.434	142				

Note. BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; BADDS-Affect = Brown ADD Scales-Affect, T Score; NEO-Neuroticism = NEO-Personality Inventory-Neuroticism-T Score.

[a]R^2 = .015, [b]R^2 = .096, [c]R^2 = .085, [d]R^2= .091

*p < .05, **p < .01, ***p < .001

The results of these post hoc ANOVAs should be interpreted with caution regarding the BDI-II and PSWQ, due to significant findings from Levene's test. Field

admits that he includes the Levene's test in his book out of expectation from others only. Field points out that Levene's test can be statistically significant in larger samples, in which there are small effects that have less practical significance. Field reports that many statisticians have discontinued using the test, specifically for two reasons, one of which is relevant in the present context. Of importance, Levene's test is less practical with larger sample sizes and when groups are not equal. This is precisely the case in the present instance. Although there are many ways of adjusting the data to resolve differences in variances, there is controversy about doing so as well. Field (2018) recommends using robust statistical analysis as the best alternative.

In line with Field (2018), because there were significant differences in variances between the diagnostic groups for the BDI-II and PSWQ, robust tests of equality of means were performed in each instance. Although the original post hoc ANOVAs between groups were significant for the BDI-II (p =.003) and PSWQ (p = .006), the results from performing a Brown-Forsythe were not significant. For the BDI-II the Brown-Forsythe statistic (3, 12.295) = 3.077, p = .067. For the PSWQ the Brown-Forsythe statistic (3, 18.841) = 2.844, p = .065. By using this more robust statistic, in each instance the results went from being statistically significant to approaching significance only. For these reasons post hoc testing with the Tukey was not performed on the BDI-II and PSWQ.

Post hoc comparisons using the Tukey HSD indicated that the mean score for the ADHD + SCT group on the BADDS-Affect scale was significantly higher than the mean BADDS-AFFECT score of those in the ADHD + subclinical SCT group (p < .01). The results of these post hoc analyses are provided in Table 6. It is important to note that the

group sizes used in these analyses are unequal. The harmonic mean of the group sizes is used, Type I error levels may be inaccurate.

Table 6

Post Hoc Comparisons of Affect Regulation Using Tukey's HSD

Dependent Variable	(I) Diagnostic group	(J) Diagnostic group	Mean Difference (I-J)	*SE*	*p*	95% CI Lower Bound	95% CI Upper Bound
Brown ADD Scales-Affect, T Score	ADHD + SCT	ADHD + subclinical SCT	8.11**	2.505	.008	1.60	14.63
		ADHD-only	6.92	2.811	.070	-.39	14.23
		SCT-only	-.93	4.875	.998	-13.61	11.75
	ADHD + subclinical SCT	ADHD + SCT	-8.11**	2.505	.008	-14.63	-1.60
		ADHD-only	-1.19	3.173	.982	-9.44	7.06
		SCT-only	-9.04	5.093	.290	-22.28	4.20
	ADHD-only	ADHD + SCT	-6.92	2.811	.070	-14.23	.39
		ADHD + subclinical SCT	1.19	3.173	.982	-7.06	9.44
		SCT-only	-7.85	5.250	.443	-21.50	5.80
	SCT-only	ADHD + SCT	.93	4.875	.998	-11.75	13.61
		ADHD + subclinical SCT	9.04	5.093	.290	-4.20	22.28
		ADHD-only	7.85	5.250	.443	-5.80	21.50

Note. The error term is *MS* (Error) = 151.994.

** $p < .01$

Hypothesis 2 Analysis

It was hypothesized that there would be a significant difference between subjects diagnosed with ADHD + SCT, ADHD + subclinical SCT, ADHD-only, and SCT only, with those diagnosed with ADHD + SCT exhibiting significantly more impaired cognitive functioning than all other groups. Impaired cognitive functioning was operationally defined as standardized scores from the Coding task and Digit Span task, as well as the BDEFS Summary score, A MANOVA was conducted to determine if the scores on the measures of cognitive functioning significantly differed between subjects who exhibited ADHD + SCT, ADHD + subclinical SCT, SCT only, or ADHD-only.

To correct for the experiment-wise error rate of three comparisons, the critical *p* value was set at 0.01. An a priori power analysis determined that a sample size of 120 would be required to achieve the minimal power level of 0.8 with $p = 0.01$, four groups, and three dependent variables. Of the total sample of 143 subjects, there were 74 in the ADHD + SCT group, 36 in the ADHD + subclinical SCT group, 24 in the ADHD-only group, and seven in the SCT-only group.

Tests of normality revealed a negative skew (-3.138) and a leptokurtic distribution (12.022) for subjects' BDEFS Summary score. Skew and kurtosis were within normal limits for subjects' scores on the Coding task and Digit Span task. Although the distribution of subjects' BDEFS Summary scores violates the assumptions of normalcy, this distribution makes sense because this is a clinical sample of individuals with ADHD, SCT, or both. These diagnoses are associated with difficulties in executive functioning. The variance-covariance ratio was tested using Box's test of equality of covariance

matrices. Box's M was calculated to be 13.316 and was found to be not significant (p =.187), indicating the assumptions of equal variance were met.

Scores from the Coding task, Digit Span task, and BDEFS Summary were tested for linearity and multicollinearity. As shown in Table 7, none of these variables were correlated at the critical significance level ($p > .01$). As per the MANOVA, there were no significant effects of diagnostic group on subjects' scores on the measures of cognitive functioning. Specifically, using Pillai's trace there was not a significant effect of the diagnostic group on measures of cognitive functioning, V = .035, $F(6, 278) = .828$, p = .55. Using Wilks' lambda, there was not a significant effect of the diagnostic group on measures of cognitive functioning, $\Lambda = .965$, $F(6, 276) = .827$, $p = .55$. Using Hotelling's trace statistic, there was not a significant effect of the diagnostic group on measures of cognitive functioning, T = .036, $F(6, 276)$, = .824, $p = .55$. Using Roy's largest root test, there was not a significant effect of the diagnostic group on measures of cognitive functioning, $\Theta = .033$ $F(3, 139) = 1.511$, $p = .214$. The measures of cognitive functioning examined did not significantly differ between subjects who exhibited varied levels of ADHD and SCT symptomatology in this analysis. Therefore, univariate tests of between subjects' effects were not interpretable.

Table 7

Correlations Between Measures of Cognitive Functioning

	BDEFS Summary	Coding task	Digit Span task
Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary	-	-.081	.146*
WAIS-IV Digit Symbol Coding, Scaled Score	-.081	-	.184*
WAIS-IV Digit Span, Scaled Score	.146*	.184*	-

Note. BDEFS Summary = Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary. Coding task = WAIS-IV Digit Symbol Coding, Scaled Score. Digit Span task = WAIS-IV Digit Span, Scaled Score.

*$p < .05$ 1-tailed

Table 8

Multivariate Tests for Differences in Cognitive Functioning Between Groups[a]

Intercept	Value	*F*	Hyp. *df*	Error *df*	*p*	Partial Eta Squared	Noncent. Parameter	Observed Power[d]
Pillai's Trace	.918	769.097[b]	2	138	.00	.918	1538.194	1.000
Wilks' Lambda	.082	769.097[b]	2	138	.00	.918	1538.194	1.000
Hotelling's Trace	11.146	769.097[b]	2	138	.00	.918	1538.194	1.000
Roy's Largest Root	11.146	769.097[b]	2	138	.00	.918	1538.194	1.000
Diagnostic group								
Pillai's Trace	.035	.828	6	278	.55	.018	4.970	.327
Wilks' Lambda	.965	.827[b]	6	276	.55	.018	4.963	.326
Hotelling's Trace	.036	.826	6	274	.55	.018	4.956	.326
Roy's Largest Root	.033	1.511[c]	3	139	.21	.032	4.534	.392

Note. Hyp. *df* = Hypothesis degrees of freedom

[a]Design: Intercept + Diagnostic group

[b]Exact statistic

[c]The statistic is an upper bound on *F* that yields a lower bound on the significance level.

[d]Computed using $\alpha = .05$

Hypothesis 3 Analysis

It was hypothesized that anxiety and deficits in executive functioning would predict SCT after accounting for ADHD symptomatology. SCT was operationally defined as subjects' percentile rank on the BAARS-IV SCT subscale (SCT Percentile Rank). Anxiety was operationalized as subjects' scores on the PSWQ. Executive functioning was operationalized as subjects' BDEFS Summary score. ADHD symptomatology was operationally defined as subjects' scores on the *DSM-IV* Inattentive symptoms and *DSM-IV* Hyperactive/Impulsive symptoms scales of the CAARS.

A regression analysis was conducted to examine the associations between internalizing symptoms (operationalized as scores on the BDI-II, PSWQ, BADDS-Affect, and NEO-Neuroticism) and cognitive functioning (operationalized as BDEFS Summary, Digit Span task, and Coding Task) on the one hand, and SCT symptomatology, on the other hand. The normality, homoscedasticity, linearity, and multicollinearity of the data were examined to test the assumptions of this regression analysis. To test the normality of the data, a frequencies distribution calculated the skew and kurtosis of the data. Subjects' SCT Percentile Ranks were found to be negatively skewed (-2.317) and leptokurtic (6.939). Subjects' BDEFS Summary scores were negatively skewed (-3.128) and leptokurtic (12.022). Bonferroni corrections were used to account for the increased Type I experiment-wise error rate caused by conducting multiple statistical tests on the same data set. To correct for the experiment-wise error rate of three comparisons, the critical *p* value was set at 0.01.

Table 9

Summary of Pearson Correlations Between Measures of SCT, Internalizing Symptoms, and Cognitive Functioning

Measure	1	2	3	4	5	6	7	8	9
1. SCT Percentile Rank	-	.11	.05	.33**	.23**	.28**	-.14*	.07	.46**
2. CAARS Inattentive T Score	.11	-	.04	.00	.01	.05	.01	-.09	.07
3. CAARS Hyperactive/Impulsive T Score	.05	.04	-	.02	.04	-.01	-.16	-.07	.15*
4. Beck Depression Inventory-II	.33**	.00	.02	-	.02	.52**	-.08	-.13	.24**
5. Penn State Worry Questionnaire	.23**	.01	.04	.02	-	.45**	-.12	.04	.42**
6. Brown ADD Scales-Affect, T Score	.28**	.05	-.01	.52**	.45**	-	-.03	-.06	.35**

Measure	1	2	3	4	5	6	7	8	9
7. WAIS-IV Coding Task, Scaled Score	-.14*	.01	-.16*	-.08	-.12	-.03	-	.18*	-.08
8. WAIS-IV Digit Span, Scaled Score	.07	- .09	-.07	-.13	.04	-.06	.18*	-	.15*
9. Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary	.46**	.07	.15*	.24**	.42**	.35**	-.08	.15*	-

Note. 1 = Barkley Adult ADHD Rating Scale: Self report Current Symptoms - sluggish cognitive tempo, Percentile Rank; 2 = Conners Adult ADHD Rating Scales-Inattentive Symptoms, T Score; 3 = Conners Adult ADHD Rating Scales-Hyperactive-Impulsive Symptoms, T Score; 4 = Beck Depression Inventory-II; 5 = Penn State Worry Questionnaire; 6 = Brown ADD Scales-Affect, T Score; 7 = WAIS-IV Digit Symbol Coding, Scaled Score; 8 = WAIS-IV Digit Span Task, Scaled Score.

*$p < .05$ one-tailed, **$p < .01$ one-tailed

The association between scores on the measures of cognitive processing and internalizing symptoms were examined using Pearson product-moment correlations to

determine which variables were to be retained for regression analysis. There was no significant relationship found between SCT Percentile Rank and scores on the CAARS inattentive subscale, $r = .111$, $p = .093$; or between SCT Percentile Rank and scores on the CAARS hyperactive/impulsive subscale, $r = .049$. $p = .280$. SCT Percentile Rank also did not evince a significant relationship with performance on the Digit Span task, $r = .066$, $p = .217$. Subjects' scores on the BDI-II, PSWQ, BADDS-Affect, Coding task, and BDEFS Summary all demonstrated significant Pearson product-moment correlations with SCT Percentile Rank (See Table 9 for details of the correlational matrix). There was a significant relationship between BDI-II scores and SCT Percentile Rank, $r = .329$, $p < .001$. There was also a significant relationship between PSWQ scores and SCT Percentile Rank, $r = .225$, $p = .003$. Scores from the BADDS-Affect scale were significantly related to SCT Percentile Rank, $r = .277$, $p = .000$. In addition, there was a significant relationship between Coding Task scores and SCT Percentile Rank, $r = -.138$, $p = .050$. Finally, there was a significant relationship between BDEFS Summary scores and SCT Percentile Rank, $r = .463$, $p < .001$.

Original statistical regression plan for hypothesis 3. The original statistical regression plan, which was not used in this study, was to enter the CAARS Inattentive and CAARS Hyperactive/Impulsive symptom scales into regression model 1 as the only predictor variables for SCT Percentile Rank. This would have been intended to examine the relationship between ADHD symptomatology and SCT and to account for the strong relationship between ADHD and SCT seen in previous research.

Regression model 2 would have retained the CAARS scores and added relevant scores from the measures of internalizing symptoms (BDI-II, PSWQ, and BADDS-

Affect) and cognitive functioning (BDEFS Summary score and the Coding task) as predictor variables for SCT Percentile Rank. Howeverbecause neither of the CAARS scores was correlated with the dependent variable in these analyses, there was no need to ensure they were entered hierarchically as the first predictors into the model. Therefore, all five remaining predictors were entered into the regression model using the forced entry method.

Statistical regression testing hypothesis 3. The regression model used in this study, including the BDI-II, PSWQ, BADDS-Affect, BDEFS Summary, and Coding Task as predictors, was found to predict a significant portion of the variance in SCT Percentile Rank scores (R = .523; R^2 = .274, p < .001). The Durbin-Watson test found the autocorrelation of the model's residuals was within an acceptable range (1.042). The adjusted coefficient of determination indicates shrinkage of only 2.6% (Adjusted R^2 = .248) if this model was generalized to the population of adults with either ADHD or SCT. Details of the model summary can be found in Table 10.

Table 10

Regression Model Summary Utilizing Measures of Internalizing Symptoms and Cognitive Functioning to Predict SCT Percentile Ranks

R	R^2	Adjusted R^2	*SE* of the Estimate	R^2 Change	*F* Change	*df*1	*df*2	Sig. *F* Change	Durbin-Watson
.523[a]	.274	.248	9.67217	.274	10.343	5	137	.000	1.042

[a]Predictors: (Constant), Barkley Deficits in Executive Functioning Scale: Self Report – Total EF Summary, WAIS-IV Digit Symbol Coding Scaled Score, Beck Depression Inventory-II, Penn State Worry Questionnaire, Brown ADD Scales-Affect T Score

As shown in Table 11, the overall regression analysis was significant, indicating the regression model predicted SCT Percentile Ranks significantly better than chance, $F(5,137) = 10.34$, $p < .001$. Two of the predictor variables were found to significantly contribute to the regression model. Scores on the BDI-II significantly predicted SCT Percentile Rank, $\beta = .215$, $t(137) = 2.514$. $p = .013$. Additionally, BDEFS Summary scores significantly predicted SCT percentile, $\beta = .403$, $t(137) = 4.921$, $p < .001$. Other predictor variables were not significantly associated with SCT Percentile Rank in the regression model. Tolerance and Variance Inflation Factor for all predictor variables were normal, indicating no significant multicollinearity. Detailed statistics for each predictor variable can be found in Table 12.

Table 11

Overall Regression Analysis With Predictor Variables to Subjects' SCT Percentile Ranks[a]

Model 1	*SS*	*df*	*MS*	*F*	*p*
Regression	4837.768	5	967.554	10.343***	.000[b]
Residual	12816.469	137	93.551		
Total	17654.237	142			

[a]Dependent Variable: Barkley Adult ADHD Rating Scale: Self report Current Symptoms - sluggish cognitive tempo, Percentile based on age and raw score.

[b]Predictors: (Constant), Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary, WAIS-IV Digit Symbol Coding Scaled Score, Beck Depression Inventory-II, Penn State Worry Questionnaire, Brown ADD Scales-Affect T Score.

***$p < .001$

Table 12

Coefficients of Predictor Variables to Subjects' SCT Percentile Ranks[a]

	Unstandardized Coefficients		Standardized Coefficients			Collinearity Statistics	
Model 1	B	*SE*	β	*t*	*p*	Tolerance	VIF
(Constant)	40.845	9.603		4.253	.000		
Beck Depression Inventory II	.220	.087	.215*	2.514	.013	.724	1.380
Penn State Worry Questionnaire	-.016	.061	-.023	-.262	.794	.716	1.397
Brown ADD Scales-Affect, T Score	.029	.081	.033	.355	.723	.609	1.642
WAIS-IV Digit Symbol Coding, Scaled Score	-.339	.278	-.090	-1.219	.225	.977	1.023
Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary	.518	.105	.403***	4.921	.000	.790	1.266

Note. VIF = Variance Inflation Factor

[a]Dependent Variable is Barkley Adult ADHD Rating Scale: Self report Current Symptoms - sluggish cognitive tempo, Percentile based on age and raw score

*$p < .05$, ***$p < .001$

Exploratory Analyses

Two exploratory analyses were conducted and are explored below.

Diagnostic group differences in executive functioning

A one-way ANOVA was conducted to explore whether subjects from the diagnostic groups, differed significantly in executive functioning, operationalized as BDEFS Summary scores. To correct for the experiment-wise error rate, the critical *p* value was set at .01. As previously mentioned, tests of normality revealed a negative skew (-3.138) and a leptokurtic distribution (12.022) for BDEFS Summary scores. This violates the assumption of normality of the data for the ANOVA.

Results of the one-way ANOVA found there was a significant difference between the four diagnostic groups on BDEFS Summary scores, $F(3,139) = 7.08$, $p < .001$. A Bonferroni corrected post hoc revealed subjects with ADHD + SCT had significantly higher mean BDEFS Summary scores than the ADHD + subclinical SCT subjects, $p = .007$, $d = 5.53$. Significant differences were also noted between ADHD + SCT subjects' higher mean BDEFS Summary scores compared to the ADHD-only subjects, $p = .001$, $d = 7.42$. No significant differences were found between the seven subjects with SCT only and the other diagnostic groups and no significant differences were found between the ADHD + subclinical SCT groups' mean BDEFS Summary scores and the mean scores of the SCT-only or ADHD-only groups. A post hoc Tukey HSD test found the same pattern of significant findings. Results of the post hoc tests are provided in Table 13 and Table 14. This exploratory analysis implies that executive functioning measures may be the best indicators of the kinds of cognitive functioning issues that differentiate ADHD and SCT

symptomatology, as opposed to tests like the Coding and Digit Span tasks from the WAIS-IV in adults with ADHD + SCT.

Table 13

Results of One-Way Analysis of Variance Examining Diagnostic Group Influence on Subjects' BDEFS Scores

	SS	*df*	*MS*	*F*	*p*
Between Groups	1416.869	3	472.290	7.084***	.000
Within Groups	9267.033	139	66.669		
Total	10683.902	142			

***$p < .001$

Table 14

Post Hoc Tukey's HSD and Bonferroni Tests Examining Difference Between Diagnostic Groups' Mean Scores on the BDEFS Summary Score

						95% CI	
	Diagnostic Group (I)	Diagnostic Group (J)	Mean Difference (I-J)	*SE*	*p*	Lower Bound	Upper Bound
Tukey's HSD	ADHD + SCT	ADHD + subclinical SCT	5.528**	1.659	.006	1.21	9.84
		ADHD-only	7.423**	1.861	.001	2.58	12.26
		SCT-only	1.429	3.229	.971	-6.97	9.82
	ADHD + subclinical SCT	ADHD + SCT	-5.528*	1.659	.006	-9.84	-1.21
		ADHD-only	1.895	2.101	.804	-3.57	7.36
		SCT-only	-4.099	3.373	.618	-12.87	4.67
	ADHD-only	ADHD + SCT	-7.423*	1.861	.001	-12.26	-2.58
		ADHD + subclinical SCT	-1.895	2.101	.804	-7.36	3.57
		SCT-only	-5.995	3.477	.315	-15.04	3.05
	SCT-only	ADHD + SCT	-1.429	3.229	.971	-9.82	6.97
		ADHD + subclinical SCT	4.099	3.373	.618	-4.67	12.87
		ADHD-only	5.995	3.477	.315	-3.05	15.04

	Diagnostic Group (I)	Diagnostic Group (J)	Mean Difference (I-J)	*SE*	*p*	95% CI Lower Bound	95% CI Upper Bound
Bonferroni	ADHD + SCT	ADHD + subclinical SCT	5.528**	1.659	.007	1.09	9.97
		ADHD-only	7.423**	1.861	.001	2.44	12.41
		SCT-only	1.429	3.229	1.00	-7.21	10.07
	ADHD + subclinical SCT	ADHD + SCT	-5.528**	1.659	.007	-9.97	-1.09
		ADHD-only	1.895	2.101	1.00	-3.73	7.52
		SCT-only	-4.099	3.373	1.00	-13.13	4.93
	ADHD-only	ADHD + SCT	-7.423**	1.861	.001	-12.41	-2.44
		ADHD + subclinical SCT	-1.895	2.101	1.00	-7.52	3.73
		SCT-only	-5.995	3.477	.521	-15.30	3.31
	SCT-only	ADHD + SCT	-1.429	3.229	1.00	-10.07	7.21
		ADHD + subclinical SCT	4.099	3.373	1.00	-4.93	13.13
		ADHD-only	5.995	3.477	.521	-3.31	15.30

*$p < .05$ **$p < .01$.

The impact of SCT in adults with ADHD on internalizing disorders and cognitive functioning. An additional exploratory MANOVA was conducted to examine whether the degree of internalizing symptoms and cognitive functioning varied in adults with ADHD who have differing levels of SCT. Diagnostic groups served as the independent variable in this analysis. The measures of internalizing symptoms and cognitive functioning that were found to significantly differ between diagnostic groups across the previous analyses in this study served as the dependent variables. The seven subjects with SCT only were not included in these analyses because they did not exhibit clinically significant levels of ADHD.

Previous analyses found subjects' BDEFS Summary scores were positively skewed and leptokurtic, with a skew of 3.128 (SE = 0.203) and kurtosis of 12.022 (SE = 0.403). Box's test found that the assumptions of homogeneity of covariance were not met between the diagnostic groups. The value of Box's M was calculated as 95.388 and was found to be significant ($p < .001$).

Levene's test indicated that the homogeneity of variance across groups was unequal. As indicated in Table 15, the variance in subjects scores was unequal between the three diagnostic groups on the BDI-II, $F(2, 133) = 6.295$, $p = 0.002$; Digit Span task, $F(2, 133) = 5.066$, $p = .008$; and BDEFS Summary scores, $F(2, 133) = 6.283$, $p = 0.002$.

Table 15

Levene's Test of Equality of Error Variances for Measures of Internalizing Symptoms and Cognitive Functioning

	F	*df*1	*df*2	*p*
NEO-Personality Inventory-Neuroticism-T Score	.983	2	133	.377
Beck Depression Inventory-II	6.295**	2	133	.002
Penn State Worry Questionnaire	1.387	2	133	.253
Brown ADD Scales-Affect, T Score	.901	2	133	.409
WAIS-IV Digit Span, Scaled Score	5.066**	2	133	.008
Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary (1-5), % score	6.283**	2	133	.002

Note. Design = Intercept + Diagnostic Group

** $p < .01$

Due to unequal sample sizes, groups differing along more than one variate, violations of normalcy of the data, and the assumptions of equal covariance; Pillai's trace was used as the significance statistic in the MANOVA because it is more robust than the other statistics and more appropriate for violations of model assumptions (Olson, 1974; Field, 2013). Pillai's trace indicated a significant overall effect of diagnostic group on measures of the following internalizing symptoms and cognitive processing, BDI-II, PSWQ, BADDS-Affect, and BDEFS Summary, V = 0.225, $F(12.\ 258) = 2.720$, $p = 0.002$. The observed power for this analysis was 0.982. Details of the MANOVA are presented in Table 16.

Table 16

Multivariate Tests for Differences in Internalizing Symptoms and Cognitive Functioning Between Adults with ADHD and Varying Levels of SCT[a]

Intercept	Value	*F*	Hyp. *df*	Error *df*	*p*	Partial Eta Squared	Noncent. Parameter	Observed Power[d]
Pillai's Trace	.992	2672.558[b]	6	128	.000	.992	16035.346	1.000
Wilks' Lambda	.008	2672.558[b]	6	128	.000	.992	16035.346	1.000
Hotelling's Trace	125.276	2672.558[b]	6	128	.000	.992	16035.346	1.000
Roy's Largest Root	125.276	2672.558[b]	6	128	.000	.992	16035.346	1.000
Diagnostic group								
Pillai's Trace	.225	2.720	12	258	.002	.112	32.643	.982
Wilks' Lambda	.781	2.810[b]	12	256	.001	.116	33.715	.986
Hotelling's Trace	.274	2.898	12	254	.001	.120	34.772	.988
Roy's Largest Root	.246	5.278[c]	6	129	.000	.197	31.670	.994

Note. Hyp. *df* = Hypothesis degrees of freedom

[a]Design: Intercept + Diagnostic group

[b]Exact statistic

[c]The statistic is an upper bound on *F* that yields a lower bound on the significance level.

[d]Computed using $\alpha = .05$

As determined by univariate ANOVA, diagnostic group had a significant effect on BDI-II, $F(2, 133) = 6.876$, $p = 0.001$; PSWQ, $F(2,133) = 5.286$, $p = 0.004$; BADDS-Affect scale, $F(2, 133) = 6.714$, $p = 0.002$; and BDEFS Summary score, $F(2, 133) = 10.343$, $p < 0.001$. However, the following were not significant: the effect of diagnostic group on NEO-Neuroticism, $F(2, 133) = 1.018$, $p = 0.364$, and the Digit Span task, $F(2, 133) = 0.543$, $p = 0.582$. Results from the univariate ANOVAs are provided in Table 17.

Table 17

Discriminant Analyses of Variance Examining Between-Subjects Effects of Diagnostic Group on Dependent Variables

Dependent Variable	Type III *SS*	*df*	*MS*	*F*	*p*	Observed Power
NEO-Personality Inventory-Neuroticism-T Score	287.762	2	143.881	1.018	.364	.225
Beck Depression Inventory-II	1342.413	2	671.207	6.876**	.001	.917
Penn State Worry Questionnaire	2509.601	2	1254.801	5.826**	.004	.865
Brown ADD Scales-Affect, T Score	1976.481	2	988.240	6.714**	.002	.911
WAIS-IV Digit Span, Scaled Score	8.287	2	4.144	.543	.582	.138
Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary	1402.799	2	701.400	10.343***	.000	.986

**$p < .01$ ***$p < .001$

Levene's test indicated the variance was unequal between the three diagnostic groups on the BDI-II, Digit Span task, and BDEFS Summary scores. Since there were significant differences in variances between the diagnostic groups for the BDI-II, Digit Span task, and BDEFS summary scores, robust tests of equality of means were performed in each instance. Brown-Forsythe's test indicated that the homogeneity of variance across groups was unequal between the three diagnostic groups on the BDI-II (2, 115.844) = 8.611, $p < .001$ and BDEFS Summary score (2, 66.89) = 7.558, $p = .001$. The assumption of homogeneity of variance was met between the groups on the Digit Span task (2, 111.652) = .663, $p = .517$.

The pattern of the results was the same for the original post hoc discriminant ANOVAs and the ANOVAs utilizing the Brown-Forsythe statistic. As determined by univariate ANOVA using the Brown-Forsythe as the *F* statistic, diagnostic group had a significant effect on BDI-II, $F(2, 133) = 6.876$, $p = 0.001$; and BDEFS Summary score, $F(2, 133) = 10.343$, $p < 0.001$. The effect of diagnostic group on the Digit Span task was not significant, $F(2, 133) = 0.543$, $p = 0.582$.

Due to unequal homogeneity of variance, the Games Howell test was used for post hoc comparisons of group means. The test compares the difference between each pair of means with appropriate adjustment for the multiple testing. Because the Games Howell post-hoc test does not assume homogeneity of variances or equal sample sizes, it is a more robust statistic when the assumption of homogeneity of variances is violated (Ruxton & Beauchamp, 2008).

Post-hoc comparisons using the Games Howell statistic indicated that the mean BDI-II score of those in the ADHD + SCT group was significantly higher than those in

the ADHD-only group ($p = 0.002$). The ADHD + SCT group's mean PSWQ score was significantly higher than both the ADHD + subclinical SCT group ($p = 0.023$) and the ADHD-only group ($p = 0.032$). The ADHD + SCT group exhibited a significantly higher mean score on the BADDS-Affect scale than the ADHD+ subclinical SCT ($p = 0.005$) and ADHD-only group ($p = 0.027$). The ADHD + SCT group had a significantly higher mean BDEFS Summary score than the ADHD + subclinical group ($p = 0.003$) and the ADHD-only group ($p < 0.001$). The three groups investigated in this analysis did not have significant differences between their mean scores on NEO-Neuroticism or the Digit Span task. Detailed results of these post hoc tests are provided in the appendix. As with previous analyses, the unequal group sizes may impact the accuracy of Type 1 error levels in these analyses.

Chapter 5: Discussion

The results of the current study indicate that there are significant differences between those diagnosed with ADHD + SCT, ADHD + subclinical SCT, and ADHD only on many of the examined measures of internalizing symptoms and cognitive functioning. Subjects' scores on measures of depression, worry, affect regulation, and executive dysfunction were found to be higher amongst the subjects diagnosed with ADHD + SCT, than those with ADHD only, or those with ADHD and subthreshold levels of SCT, in many analyses. The handful of subjects in the SCT-only group did not significantly differ from other groups on these measures. Measures of cognitive processing speed, auditory working memory, and neuroticism did not differ between groups.

The results of the analyses partially supported the first hypothesis that there would be significant differences between the four diagnostic groups on measures of internalizing symptoms. Similar to previous studies examining internalizing symptoms in individuals with varying levels of ADHD and SCT symptomatology, the ADHD + SCT group exhibited significantly higher mean scores on a self-report measure of affect regulation than the ADHD + subclinical SCT group. Notably, these differences were not evident in all of the diagnostic groups, in this or previous studies (Carlson & Mann, 2002; Kamradt et al., 2018). On the other hand, the diagnostic groups did not differ on measures of depression, anxiety, or neuroticism.

Findings from this study did not support the second hypothesis, which posited diagnostic groups would significantly differ on measures of cognitive functioning, which was operationalized as measures of executive functioning, cognitive processing speed,

and auditory working memory. None of these variables were found to significantly differ between the diagnostic groups. Previous research produced mixed results in terms of significance and effect size when investigating the relationship between SCT and measures of executive functioning (Barkley, 2012; Bauermeister et al., 2012; Jarrett et al., 2017; Leikauf and Solanto, 2017), as well as SCT and measures of neuropsychological functioning (Jacobsen et al., 2018; Bauermeister et al., 2012). This pattern of inconsistent findings in the literature and this study may be attributable to diffuse deficits in cognitive functioning for those with ADHD and SCT, rather than exhibiting discrete and easily identifiable areas of impairment (Hervey et al., 2014; Mapou, 2019). This pattern of results in our sample may have been related to a relatively higher level of functioning in this sample, as compared to the general adult ADHD population (more on this below).

This study also found symptoms of impaired executive functioning and depression significantly predicted SCT symptomatology in adults who exhibit clinical levels of ADHD or SCT symptoms. This was partially consistent with the third hypothesis, which further predicted anxiety and executive functioning would be associated with SCT, after accounting for ADHD symptomatology. These findings are consistent with previous lines of research that have shown SCT symptomatology significantly contributes to the variance in internalizing symptomatology, above and beyond the contributions of ADHD symptoms (Becker, Langberg, et al., 2014). Depression may be a better predictor of SCT symptomatology than anxiety, not just due to similarities in their symptom presentations, but from common underlying symptoms, which overlap with feeling tired and lethargic, being in a fog, being underactive or slow

moving, and feeling apathetic or unmotivated in SCT (Becker, Marshall, & McBurnett, 2014).

Interestingly, ADHD symptomatology was not associated with SCT symptomatology in this sample, which is directly contrary to the well-established relationship between inattentive symptoms of ADHD and SCT symptomatology (Barkley, 2011a, 2012; Becker & Barkley, 2018; Becker et al., 2016; Garner et al., 2010; Leikauf & Solanto, 2017; Lunsford-Avery et al., 2018; Wåhlstedt & Bohlin, 2010). Nonetheless, this finding is consistent with prior research demonstrating inattentive, hyperactive/impulsive, and SCT symptoms are unique from one another and represent unique symptom domains that frequently occur together (Bauermeister et al., 2012; Becker, Marshall, & McBurnett, 2014; Neeper & Lahey, 1986).

Interpretations and Implications

Differences in Internalizing Symptoms Between Diagnostic Groups

The current study explored the differences in internalizing symptoms; such as anxiety, depression, affect regulation, and neuroticism; between individuals with varying levels of ADHD and SCT symptomatology. The results of the analyses were partially consistent with the first hypothesis' prediction that there would be a significant difference between subjects across the diagnostic groups, with those diagnosed with ADHD + SCT having higher levels of internalizing symptoms than the other diagnostic groups. The first hypothesis was not supported, in that individuals who exhibited clinical levels of both ADHD and SCT symptomatology only differed from one other group on one measure of internalizing symptoms in this study. Those with both clinical ADHD and SCT symptomatology did not report higher levels of depression than subjects with other

presentations of ADHD and SCT. Depression was operationalized as scores on the BDI-II. Although the original post hoc comparison was significant, the use of more robust post hoc tests found the differences between diagnostic groups' depression levels were not significant, but did approach significance. This is inconsistent with many previous studies that found relationships between SCT symptoms and depressive symptoms after controlling for ADHD symptoms. Furthermore, previous studies show individuals with higher levels of SCT exhibit more depressive symptoms than those with lower levels of SCT (Becker & Barkley, 2018; Bauermeister et al., 2012; Carlson & Mann, 2002; Kamradt et al., 2018; Leikauf & Solanto, 2017). However, in the exploratory MANOVA including both relevant internalizing symptoms and cognitive functioning, the ADHD + SCT subjects were found to have higher levels of depression, than those with ADHD and subclinical or negligent levels of SCT.

Subjects with ADHD + SCT did not endorse greater frequency and intensity of anxiety than the other diagnostic groups. Anxiety was operationalized as scores on the PSWQ. Again, although the original post hoc comparison of diagnostic groups' anxiety levels was significant, the use of robust post hoc tests found the between groups differences were not significant. This is inconsistent with previous research findings that higher levels of SCT are associated with greater intensity and frequency of anxiety symptoms (Bauermeister et al., 2012; Carlson & Mann, 2002; Kamradt et al., 2018; Leikauf & Solanto, 2017). The exploratory MANOVA including relevant internalizing symptoms and cognitive functioning found the ADHD + SCT subjects reported higher levels of anxiety than those with ADHD and subclinical or negligent levels of SCT

The subjects' ability to manage affect and emotion that could interfere with work was also investigated and was operationalized as scores on the affect scale of the BADDS. Subjects with both ADHD + SCT evidenced greater difficulty with affect regulation than those with ADHD + subclinical SCT. Affect regulation did not significantly differ between ADHD + SCT and ADHD-only subjects. However, in the exploratory MANOVA including both relevant internalizing symptoms and cognitive functioning, the ADHD + SCT subjects were found to have greater difficulty with affect regulation than the ADHD-only subjects. This change in significance occurred despite the difference in mean scores between these two groups remaining constant between the two analyses.

These findings imply that the SCT symptomatology may be related to challenges with depression, anxiety, and affect regulation in adults with ADHD. One possible explanation for this relationship is the similarity between the symptoms investigated. Theories attempting to explain the close relationship between anxiety, depression, and SCT posits these constructs share several core features common to internalizing disorders, such as apathy, rumination, daydreaming, inactivity, and decreased effort (Smith & Langberg, 2017).

There are also similarities in the symptoms investigated by the measures used to operationalize SCT and depression. Both the SCT scale of the BAARS-IV and the BDI-II have items pertaining to loss of energy, fatigue, and difficulty concentrating (Barkley, 2011a; Beck et al., 1996). A number of studies have demonstrated that SCT is a distinct syndrome from ADHD and internalizing disorders (Barkley, 2011a, 2012; Becker et al., 2016; Garner et al., 2010; Wåhlstedt & Bohlin, 2010). The findings of this study lend

credence to the theory that the strong relationship between internalizing symptoms and SCT may be attributable to a common etiology, rather than a simple overlap in nosology.

Neuroticism, as measured by the NEO-PI-R, was not found to be meaningfully related to diagnostic group membership or SCT symptomatology in the analyses conducted. This contrasts with previous studies that have found relationships between neuroticism, ADHD symptomatology, and SCT (Becker et al., 2018; Serine, 2016). Neuroticism has been found to be a common element among internalizing disorders, and has demonstrated significant correlations with internalizing symptoms (Griffith et al., 2010). The univariate ANOVAs that found the nonsignificant effects of diagnostic group on neuroticism were very underpowered in both the planned MANOVA ($\alpha = .01$, $1\text{-}\beta = .068$) and exploratory MANOVA ($\alpha = .05$, $1\text{-}\beta = .225$). This decreased the likelihood that a true effect could be detected, if one was present.

Differences in Cognitive Functioning Between Diagnostic Groups

The current study explored the differences in cognitive functioning, utilizing measures of cognitive processing speed, auditory working memory, and deficits in executive functioning. Interestingly, subjects' scores on the measures of cognitive functioning used in the MANOVA were not correlated with one another, so the analysis could not be interpreted. Contrary to the second hypothesis, neither subjects' auditory working memory nor their cognitive processing speed abilities were meaningfully related to SCT symptomatology or diagnostic group across these analyses. This contrasts with previous research that has found low to moderate effect sizes when examining the relationship between subtests measuring working memory and processing speed from the

WAIS and ADHD or SCT symptomatology (Hervy et al., 2004; Jacobson et al., 2018; Wechsler, 2008).

The lack of significant correlations may be attributable to the variety in constructs operationalized by the measures. Although processing speed, auditory working memory, and executive functioning are all meaningful pieces of an individual's cognitive abilities, they may be too disparate from one another to be conceptualized as parts of a similar group of cognitive functions. Similar issues have arisen in past research that used loosely related aspects of cognitive functioning to create a new composite score for use in statistical analysis (Bauermeister et al., 2012).

Slow or "sluggish" processing speed is considered a key characteristic feature of SCT (Becker & Barkley, 2018). The lack of significant relationships between subjects' SCT symptoms and their performance on the Coding task of the WAIS-IV in this study was unexpected. Previous research has found limited utility in using neuropsychological measures of processing speed or working memory in diagnosing ADHD and SCT. Diffuse deficits across multiple neuropsychological and cognitive domains in this population may lead to inconsistent findings across studies examining this population (Barkley, 2019; Hervey et al., 2004; Mapou, 2019). The false negative rate of most neuropsychological measures is too high to be used as diagnostic measures for ADHD (Barkley, 2019; Matier-Sharma et al., 1995; Ramsay, 2015). Perhaps these limitations are also true for using neuropsychological measures to diagnose and measure the symptoms of SCT.

Recent research found preliminary support for the idea that SCT symptoms in adolescents are more clearly associated with measures of processing speed as the motor

demands of the measure increase. Measures of processing speed that have high motor demands, such as the grooved pegboard test, evince stronger relationships with SCT, whereas test with moderate to low motor demands, such as the Coding task, exhibit weaker and inconsistent relationships with SCT (Becker et al., 2020). Another study found many commonly used measures of processing speed also require an individual to tap into executive functioning abilities such as goal maintenance or decision making. These unintentional demands from the measure cause executive functioning abilities to contribute to the subject's performance on the measure. These additional test demands could potentially taint the operationalization of processing speed in research utilizing these measures and may have resulted in an over estimation of processing speed's relationships with other constructs in prior research (Cepeda et al.,, 2013).

Use of these measures may have contributed to the variation in significance and effect size in research examining SCT, ADHD, and processing speed, because the measures of processing speed utilized may be tapping into an unrelated constructs such as motor control, or constructs highly related to ADHD and SCT symptoms such as executive functioning. Future research would benefit from identification and utilization of measures that are relatively pure measures of processing speed. These would demonstrate good convergent validity with other measures of processing speed as well as discriminant validity from adjacent cognitive abilities like executive functioning.

Clinically, neuropsychological measures provide valuable information on cognitive deficits, and are useful for identifying targets for intervention and treatment planning, but may not be ideal for examining the deficits in individuals with ADHD or SCT in research settings (Mapou, 2019). Clinicians and researchers advocate for more

ecologically valid tools with better predictive power for identifying the difficulties in cognitive functioning those with ADHD and SCT face. These include clinical interviews or self-report measures of executive functioning, such as the BDEFS utilized in this study (Barkley 2011b, 2019; Barkley & Fischer, 2011; Pettersson et al., 2018).

Executive dysfunction, operationalized as the BDEFS Summary score, was strongly associated with SCT symptomatology but varied between diagnostic groups. In fact, both the exploratory ANOVA and exploratory MANOVA found that BDEFS Summary scores significantly varied between diagnostic groups. Specifically, the ADHD + SCT group exhibited greater deficits in executive functioning than the ADHD + subclinical SCT and ADHD-only groups. Executive functioning did not significantly differ between the ADHD + subclinical SCT and ADHD-only groups. Although not part of the planned analyses testing the hypotheses for this study, these findings lend some support to the second hypothesis, that the ADHD + SCT group would exhibit significantly more impaired cognitive functioning than other diagnostic groups. These results are consistent with prior research findings indicating differing patterns of executive functioning in adults with ADHD and SCT (Barkley, 2011b, 2012; Willcutt et al., 2005).

Predicting SCT Symptoms Based on Internalizing Symptoms, Cognitive Functioning, and ADHD Symptomatology

The regression analysis examined which co-occurring internalizing symptoms and aspects of cognitive functioning were the most predictive of SCT in this sample. Depression and executive functioning emerged as the only significant predictors of SCT symptoms of the eight constructs investigated. Neither of the ADHD symptom scales

from the CAARS measure was correlated with SCT symptomatology in this analysis. This is partially consistent with the third hypothesis, that anxiety and deficits in executive functioning would predict subjects' levels of SCT after accounting for ADHD symptomatology.

The lack of a correlation between ADHD and SCT symptoms was unexpectedbecause SCT symptoms, operationalized as the BAARS-IV SCT subscale, have been found to significantly correlate with inattentive symptoms, operationalized as the CAARS, in prior studies. Moreover, SCT shares as much as 50% of its variance with inattentive symptoms and up to 25% of its variance with hyperactive/impulsive symptoms (Becker & Barkley, 2018; Garner et al., 2010; Leikauf & Solanto, 2017; Wåhlstedt, & Bohlin, 2010). ADHD and SCT commonly co occur in the general population, and individuals diagnosed with ADHD + SCT made up over half of the subjects in this study. However, this surprising finding is consistent with prior research conducted to validate SCT as a separate issue from ADHD. These studies demonstrated the symptoms of SCT align with a different factor than the symptoms of ADHD (Barkley, 2011a; Bauermeister et al., 2012; Becker, Marshall, & McBurnett, 2014; Neeper & Lahey, 1986).

Anxiety was hypothesized to be the internalizing symptom that would have the most influence on SCT, due to similarities between rumination and avoidance in anxiety disorders and the daydreaming and low initiation aspects of SCT (Jacobson et al., 2018). However depressive symptoms, operationalized as subjects' scores on the BDI-II, emerged as the only internalizing symptom that was a significant contributor to a

regression model predicting SCT symptom levels (standardized beta coefficient = .215, $p = .013$).

This finding is consistent with a previous study that explored the utility of adding SCT as a predictor variable to multiple regression models attempting to predict depression and anxiety. Adding SCT to the model reduced the predictive utility of measures of ADHD symptoms and mental health treatment usage to nonsignificance, and notably increased the amount of variance explained by the models (Becker, Langberg, et al., 2014). These findings, combined with the present finding that depressive symptoms significantly predict SCT, supports the idea that the symptoms of depression and SCT have a reciprocal relationship, independent of their associations with ADHD symptomatology. These findings also imply that the feelings of apathy, lethargy, and psychomotor retardation common to depression and SCT are not merely superficially similar, but stem from a common underlying problem or factor.

Executive functioning emerged as the strongest predictor of SCT symptoms in the regression model (standardized beta coefficient = .403, $p < .001$). This was consistent with the third hypothesis' prediction that executive functioning would predict subjects' levels of SCT in the analysis. Several studies have established executive functioning is a better predictor of SCT symptoms than ADHD symptoms or internalizing symptoms, even when controlling for the relationship between executive functioning and other predictor variables (Flannery et al., 2016; Jarrett et al., 2017; Leikauf & Solanto, 2017).

For clinical practice, these results indicate the need for pretreatment screening for SCT and executive dysfunction in individuals with major depressive disorder and other mood disorders. Individuals with depression often experience symptoms that cause

impairment in their ability to function such as anhedonia, fatigue, difficulty thinking or concentrating, insomnia/hypersomnia, and inappropriate feelings of worthlessness or guilt. SCT may be contributing to or maintaining these symptoms, leading to greater impairment or interference with treatment efforts to manage depression or other mood symptoms. Individuals experiencing depression and SCT may benefit from the introduction of interventions that are typically used with individuals diagnosed with ADHD. These interventions could enhance motivation, manage fatigue, and interrupt rumination and daydreaming that occur in both SCT and depression.

The ADHD + SCT group exhibited greater levels of depression, anxiety, emotion dysregulation, and executive dysfunction than the ADHD + subclinical SCT or ADHD-only groups in the exploratory analyses. Measures of depression and executive dysfunction were found to be significant predictors of subjects' SCT symptomatology, whereas ADHD symptomatology was unrelated to SCT in this sample. This and other studies have demonstrated that ADHD and SCT are distinct psychological constructs, SCT is associated with internalizing symptomatology, and depression and executive functioning play a stronger role in the presence of SCT than ADHD (Bauermeister et al., 2012; Becker, Langberg, et al., 2014; Carlson & Mann, 2002; Flannery et al.,, 2016; Jarrett et al., 2017; Kamradt et al., 2018; Leikauf & Solanto, 2017).

Limitations

Limitations of the data set. This study utilized an existing data set. As a result, there were some artifacts that limited the analyses conducted and the generalizability of conclusions drawn from them. Across the analyses in this study, there were unequal sample sizes, violations of the assumptions of covariance, violation of the assumptions of

equal variance, as well as skewed and leptokurtic data for some measures. These limitations of the data may have impacted Type 1 error levels of the analyses conducted and limited the generalizability of the findings.

The data was gathered from new clients during intake at a university-based, adult ADHD outpatient specialty clinic in a large urban area. Individuals with clinical levels of both ADHD and SCT symptomatology are overrepresented in the sample, whereas individuals with SCT only are underrepresented, leading to unequal group sizes. Subjects in the SCT-only group had one significant between-groups difference across all of the analyses. It is possible that the very small sample size of SCT-only subjects was insufficient to provide enough power to detect meaningful differences with other groups.

The proportion of subjects in this sample with clinical levels of SCT only was comparable to that found in previous research. The prevalence of SCT in the adult population is difficult to determine because of the close relationship between ADHD, SCT, and other mental health symptomatology, as well as the status of SCT as a newly identified and less well-defined mental health construct (Barkley, 2012; Becker & Barkley, 2018; Lunsford-Avery & Mitchell, 2018). Barkley's (2012) research validating the BAARS-IV SCT subscale found that 5.8% of sampled adults endorsed five or more symptoms on the SCT subscale, indicating clinical levels of SCT. Furthermore, of the sample of 1,249 participants in Barkley's study, 72 (5.76%) met criteria for SCT, with 33 (2.64%) meeting criteria for SCT but not ADHD (Barkley, 2012). Of the participants in the present study, only 7 (4.9%) adults met criteria for SCT only. It is possible the number of subjects with SCT only in this sample is representative of a low prevalence of clinical levels of SCT without ADHD in the adult population.

The imbalance in group sizes and negligible number of SCT-only subjects may also be attributable to sampling bias for the clinic where the data were collected. New clients who present for an intake at the outpatient specialty treatment clinic are likely pursuing treatment for ADHD symptoms and co-occurring disorders. ADHD and SCT frequently co-occur. Although seeking treatment for SCT symptoms at an ADHD specialty clinic seems natural for those well versed in neurodevelopmental disorders or executive dysfunction, a layperson who is experiencing only the symptoms of SCT may not identify feelings of apathy, mental fogginess, and slow processing speed as a developmental disorder related to ADHD. This would reduce the likelihood that they would present for treatment at an adult ADHD outpatient specialty clinic and could potentially lead to sampling bias for individuals with higher levels of ADHD symptoms and bias against those with SCT only.

One critique secondary data analysis research is that the conclusions drawn from these studies are limited to the samples from which they are drawn. These findings may not be generalizable to larger populations or may be valid only for groups adequately represented in the sample (Barkley, 2019). Of the 143 subjects in this study, 93 identified as male and 111 were Caucasian. Ages and education level were both normally distributed. Subjects' age approached positive skewness (.91), indicating many subjects were on the younger side of the age distribution. Mean education level was 15.74 years, with a mode of 16. Only 16 of the 143 subjects reported having a high school education or less.

The clinic at which the data were collected is within close proximity to several large colleges and universities. Although information about subjects' student status was

not retained for the data used in this analysis, the ages and education levels in the sample may reflect a sampling bias for individuals who either attend or work for these academic institutions. As a result, the findings of this study may better reflect symptomatology in young Caucasian men with clinical levels of ADHD and SCT who have completed or are currently enrolled in a college degree program, rather than the population of adults with ADHD.

Previous research on malingering and ADHD assessment has shown that college students may over report their ADHD symptoms for secondary gain (Sollman et al., 2010). The use of self-report measures may have also led to underreporting of mental health symptoms. Young adults with ADHD tend to underreport symptoms of ADHD and other mental health disorders, due to a lack of insight about their symptoms (Barkley et al., 2008).

Although many of the subjects had an initial diagnosis of ADHD from the intake process, some may have received treatment for ADHD, internalizing disorders, or another mental health issue prior to or concurrently with the evaluation. Subjects who have benefitted from this treatment may exhibit fewer symptoms of ADHD, lower levels of internalizing symptoms, or less impairment in executive functioning than those who have never received treatment (Kessler et al., 2006). The data set did not contain information on subjects' treatment history, so there is no way to control for potential confounds.

Another limitation of this study lies in its use of the Coding task and the Digit Span task to operationalize cognitive functioning. Although these subtests from the WAIS-IV have established validity as measures of processing speed and auditory working memory, the operationalization of these measures could have been improved if

the subjects had been administered all of the subtests from the standard WAIS-IV battery. This would have allowed the study to utilize the PSI and working memory index (WMI). The WMI and PSI are each calculated from three different WAIS-IV subtests, whereas only two relevant subtests were administered as part of the diagnostic battery at the university-based, adult ADHD outpatient specialty clinic. Therefore, this study was only able to utilize only one subtest from the WMI and one from the PSI as opposed to three from each.

Limitations of the analyses. In the MANOVA testing hypothesis 1 and in the exploratory MANOVA, Levene's test indicated the variance was unequal between the four diagnostic groups on their BDI-II, PSWQ, Digit Span, and BDEFS Summary scores. Because the size of the diagnostic groups was unequal and there was heterogeneity in the variance of the groups, the accuracy of the *F* ratio may have been impacted, leading to altered Type 1 error rates. Robust post hoc ANOVAs using the Brown-Forsythe statistic caused the between groups differences on the BDI-II and PSWQ to decrease from statistical significance to approaching significance only.

Follow-up discriminant analyses to the first MANOVA utilizing ANOVAs were slightly under the desired power level of .80 for subjects' scores on the BDI-II ($1-\beta$ = .751), PSWQ ($1-\beta$ = .670), and BADDS-Affect ($1-\beta$ =.715). NEO-neuroticism was very underpowered in this analysis ($1-\beta$ =.068). Discriminant analyses to the exploratory MANOVA utilizing ANOVAs were underpowered for the Digit Span task ($1-\beta$ = .138) and again for NEO-neuroticism, ($1-\beta$ = .225). These underpowered analyses may have led to increased Type II error rates and could have contributed to the nonsignificant findings for these specific analyses (Field, 2013).

Several variables used in the analyses demonstrated troublesome levels of skew and kurtosis. Subjects' SCT Percentile Ranks were found to be negatively skewed (-2.317) and leptokurtic (6.939). BDEFS Summary scores were positively skewed (3.128) and leptokurtic (12.022). CAARS Inattentive symptom scores were negligibly negatively skewed (-2.054) and leptokurtic (6.847). Although skewed and leptokurtic scores on measures of SCT, ADHD, and executive dysfunction would be expected in a clinical sample of individuals diagnosed with ADHD and SCT, these abnormal distributions violated the assumption of normalcy of the data.

Due to unequal sample sizes, groups differing along more than one variate, and violations of normalcy of the data, Pillai's trace was used as the significance statistic for the MANOVAs because it is more robust than the other statistics to violations of model assumptions (Olson, 1974; Field, 2013). The Games Howell statistic and Brown-Forsythe statistic were used for post hoc comparisons of groups because they are more robust statistics when the assumption of homogeneity of covariance is violated. Although steps were taken to accommodate violations of normalcy in these analyses, findings should be interpreted with caution. These test statistics are robust to violations of test and data assumptions, but not immune to error or bias.

Future Directions

More research is needed to examine how SCT relates to ADHD in various populations of adults. Most of the studies reviewed were conducted on schoolchildren or undergraduate students. Few studies have examined measures of SCT in adult populations or in clinical settings (Lunsford-Avery, 2018). This study was also limited by an overrepresentation of individuals with 16 years or more of education, Caucasians, and

males. Future research into SCT should utilize both clinical and nonclinical samples from a variety of adult populations, including samples from community-based mental health clinics to access more diversity in participant demographics. Presentations of SCT in undersampled populations, such as ethnic and cultural minority groups or individuals with learning disabilities, should be explored to determine if SCT is overrepresented or underrepresented in these groups.

The use of exisiting data for these analyses resulted in unequal sizes in diagnostic groups and a dearth of subjects with SCT only. Future studies would benefit from recruiting and individuals with the express purpose of maintaining approximately equal diagnostic group sizes. This would potentially reduce error caused by unequal groups and would allow for a clearer investigation of the profiles of individuals with SCT only.

A uniformly agreed-upon set of criteria to diagnose SCT does not currently exist. By better understanding the etiology of SCT as well as the symptoms and disorders that commonly co-occur with SCT, it would be possible to formally delineate diagnostic criteria or identify key symptoms that can be used to diagnose and identify SCT.

Barkley found that SCT symptoms contributed the majority of the variance to different subscales of the BDEFS. Future research could expand upon this by examining the differences among diagnostic groups across the five subscales of the BDEFS or other valid measures of discrete executive functions. The close relationship of SCT with depression suggests a common underlying factor. Diagnostic groups of subjects with varying levels of ADHD and current major depressive disorder could be compared with ADHD-only subjects to explore the differences in SCT symptomatology. If SCT and

depression share a common factor, such as internalization, then the groups with higher levels of depression would have higher scores on an SCT measure.

In sum, these findings contribute to the research on the etiology and co-occurring mental health symptoms in adults with ADHD and SCT. This study's findings indicate internalizing symptoms and executive dysfunction are greater in those with clinically significant levels of ADHD and SCT. SCT, depression, and executive dysfunction had closer relationships to one another than ADHD symptoms in a sample of adults diagnosed with ADHD. Perhaps SCT symptomatology reflects a place in psychological pathology where "the boundaries between disorders are more porous than originally perceived" (American Psychiatric Association, 2013, p. 6).

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Appendix

Post Hoc Comparisons of Diagnostic Groups Using the Games-Howell Test

Dependent Variable	(I) Diagnostic group	(J) Diagnostic group	Mean Difference (I-J)	*SE*	*p*	95% CI Lower Bound	95% CI Upper Bound
NEO-Personality Inventory-Neuroticism-T Score	ADHD + SCT	ADHD + subclinical SCT	2.29	2.247	.567	-3.07	7.65
		ADHD-only	3.51	2.765	.419	-3.18	10.21
	ADHD + subclinical SCT	ADHD + SCT	-2.29	2.247	.567	-7.65	3.07
		ADHD-only	1.22	2.901	.907	-5.79	8.24
	ADHD-only	ADHD + SCT	-3.51	2.765	.419	-10.21	3.18
		ADHD + subclinical SCT	-1.22	2.901	..907	-8.24	5.79
Beck Depression Inventory-II	ADHD + SCT	ADHD + subclinical SCT	4.24	1.938	.079	-.38	8.86
		ADHD-only	7.96***	1.942	.000	3.30	12.61
	ADHD + subclinical SCT	ADHD + SCT	-4.24	1.938	.079	-8.86	.38
		ADHD-only	3.72	2.051	.175	-1.22	8.65
	ADHD-only	ADHD + SCT	-7.96***	1.942	.000	-12.61	-3.30
		ADHD + subclinical SCT	-3.72	2.051	.175	-8.65	1.22

Dependent Variable	(I) Diagnostic group	(J) Diagnostic group	Mean Difference (I-J)	*SE*	*p*	95% CI Lower Bound	95% CI Upper Bound
Penn State Worry Questionnaire	ADHD + SCT	ADHD + subclinical SCT	7.89*	2.915	.023	.91	14.87
		ADHD-only	9.48*	3.607	.032	.70	18.27
	ADHD + subclinical SCT	ADHD + SCT	-7.89*	2.915	.023	-14.87	-.91
		ADHD-only	1.59	4.010	.917	-8.09	11.28
	ADHD-only	ADHD + SCT	-9.48*	3.607	.032	-18.27	-.70
		ADHD + subclinical SCT	-1.59	4.010	.917	-11.28	8.09
Brown ADD Scales-Affect, T Score	ADHD + SCT	ADHD + subclinical SCT	8.11**	2.480	.005	2.18	14.04
		ADHD-only	6.92*	2.591	.027	.66	13.18
	ADHD + subclinical SCT	ADHD + SCT	-8.11**	2.480	.005	-14.04	-2.18
		ADHD-only	-1.19	2.930	.914	-8.24	5.86
	ADHD-only	ADHD + SCT	-6.92*	2.591	.027	-13.18	-.66
		ADHD + subclinical SCT	1.19	2.930	.914	-5.86	8.24

Dependent Variable	(I) Diagnostic group	(J) Diagnostic group	Mean Difference (I-J)	*SE*	*p*	95% CI Lower Bound	95% CI Upper Bound
WAIS-IV Digit Span task, Scaled Score	ADHD + SCT	ADHD + subclinical SCT	.49	.566	.659	-.86	1.85
		ADHD-only	.50	.522	.609	-.75	1.75
	ADHD + subclinical SCT	ADHD + SCT	-.49	.566	.659	-1.85	.86
		ADHD-only	.00	.587	1.000	-1.41	1.41
	ADHD-only	ADHD + SCT	-.50	.522	.609	-1.75	.75
		ADHD + subclinical SCT	.00	.587	1.000	-1.41	1.41
Barkley Deficits in Executive Functioning Scale: Self Report - Total EF Summary score	ADHD + SCT	ADHD + subclinical SCT	5.53*	2.126	.034	.36	10.70
		ADHD-only	7.42**	1.831	.001	2.91	11.93
	ADHD + subclinical SCT	ADHD + SCT	-5.53*	2.126	.034	-10.70	-.36
		ADHD-only	1.90	2.686	.761	-4.56	8.35
	ADHD-only	ADHD + SCT	-7.42*	1.831	.001	-11.93	-2.91
		ADHD + subclinical SCT	-1.90	2.686	.761	-8.35	4.56